USE OF SOFA (SEQUENTIAL ORGAN FAILURE ASSESSMENT) SCORING IN ASSESSING THE INCIDENCE AND SEVERITY OF ORGAN DYSFUNCTION AND PREDICTING THE OUTCOME IN PATIENTS WITH SEPSIS IN SURGICAL UNIT

Dissertation Submitted in the

In partial fulfillment of the regulations required for the award of

M.S. DEGREE

in

GENERAL SURGERY

THE TAMILNADU

DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI

APRIL 2016

CERTIFICATE

This is to certify that this dissertation titled **"**USE OF SOFA (SEQUENTIAL ORGAN FAILURE ASSESSMENT) SCORING IN ASSESSING THE INCIDENCE AND SEVERITY OF ORGAN DYSFUNCTION AND PREDICTING THE OUTCOME IN PATIENTS WITH SEPSIS IN SURGICAL UNIT**"** is the bonafide work of Dr. Sharath CT postgraduate student in M.S. General Surgery, Coimbatore Medical College and Hospital, Coimbatore. This study was undertaken in the department of General Surgery, Coimbatore Medical College and Hospital during the period September 2014 to September 2015 in partial fulfillment of the requirement of the "The Tamil Nadu Dr.M.G.R.Medical university" for the award of M.S. Degree in General Surgery. This dissertation has not been submitted in part or fully to any other University or Board. It gives me great pleasure to forward this dissertation.

Dr. Edwin Joe MD., BL.

The Dean,

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DECLARATION

The dissertation titled "USE OF SOFA (SEQUENTIAL ORGAN FAILURE ASSESSMENT) SCORING IN ASSESSING THE INCIDENCE AND SEVERITY OF ORGAN DYSFUNCTION AND PREDICTING THE OUTCOME IN PATIENTS WITH SEPSIS IN SURGICAL UNIT" is being submitted by me to "The Tamil Nadu Dr.M.G.R. medical university" in partial fulfillment of the regulation for the completion of the M.S. General Surgery degree examination to be held in 2016.This work has been carried out in the Department of General Surgery, Coimbatore Medical College and Hospital, Coimbatore under the guidance of Dr. S.Saradha, M. S, F.I.C.S, F.A.I.S, Professor of General Surgery, Coimbatore Medical College and Hospital, Coimbatore.

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College : Coimbatore Medical college

Dissertation Topic : Use of SOFA scoring in assessing the incidence & severity of organ dysfunction & predicting the outcome in patients with surgical sepsis.

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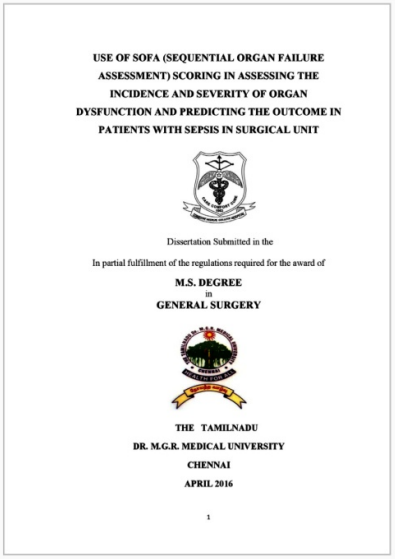

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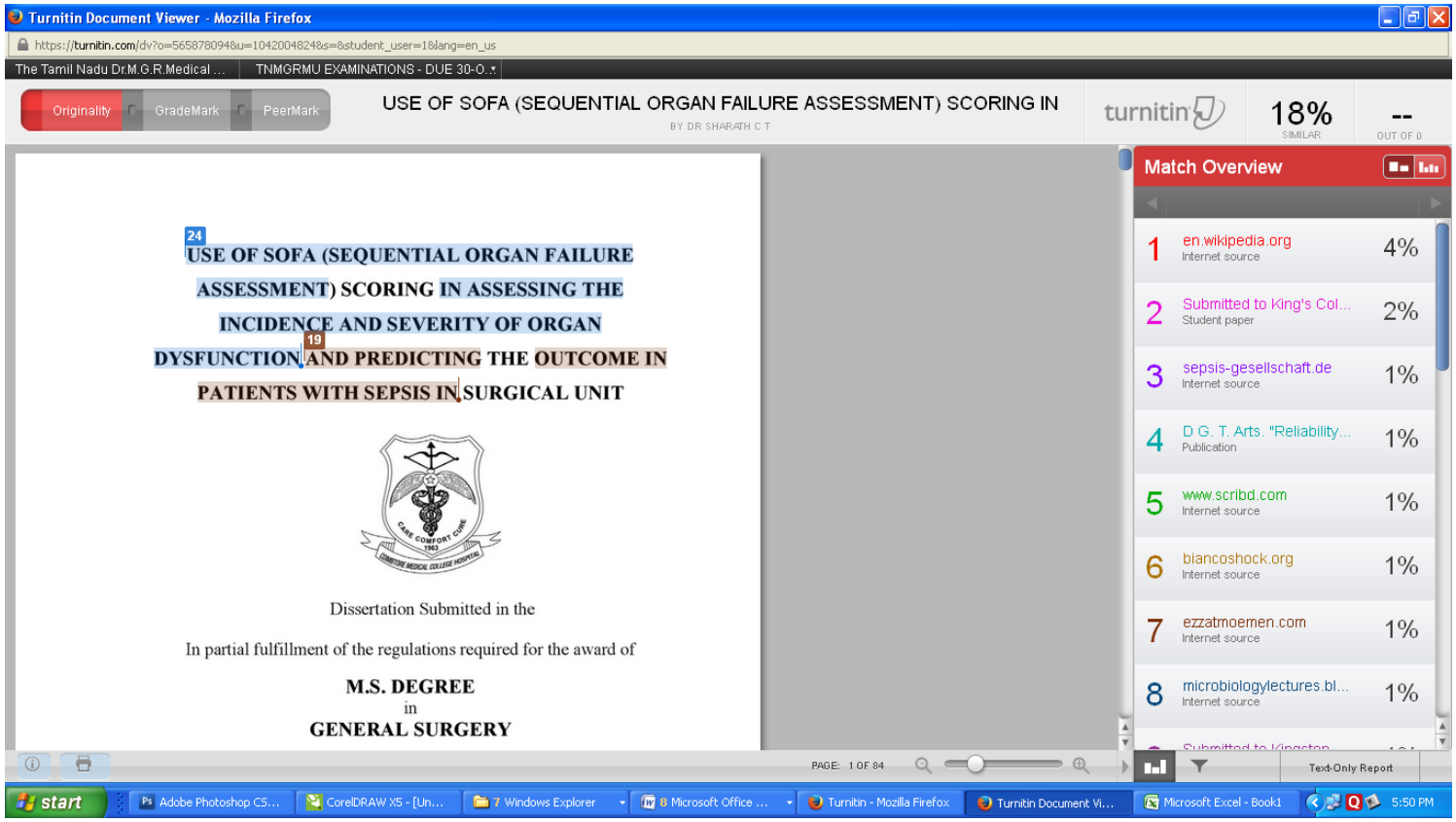
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Date :

Place : Coimbatore **Dr. Sharath C T**

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INTRODUCTION

Multi - organ dysfunction syndrome (MODS) is the leading cause of morbidity and mortality for patients admitted with sepsis, and develops in about 15% of all admissions. Over the past years many scoring models have been developed to describe the severity of illness in patient admitted with sepsis. As an example, the first Sepsis-related Organ Failure Assessment score, later called the Sequential Organ Failure Assessment (SOFA) score, was introduced in 1994[(1)].The aim was to quantify the severity of the patient's illness based on the degree of organ dysfunction, serially over time. Although severity of illness scoring systems such as the Acute Physiology and Chronic Health Evaluation (APACHE) II and the Simplified Acute Physiology Score (SAPS) II are based on the first 24hrs of admission, the SOFA scoring system takes into account the time course of a patient's condition during the entire stay in the hospital . This enables surgeons to follow the evolving disease process.

The Sequential Organ Failure Assessment (SOFA) score is a simple and objective score that allows for calculation of both the number and the severity of organ dysfunction in six organ systems. It is a six-organ dysfunction score measuring multiple organ failure daily. Each organ is graded from 0 (normal) to 4 (the most abnormal).

Although SOFA was developed primarily to describe and quantify organ function, it has been demonstrated in several studies to predict mortality and morbidity of critically ill patients. Early prediction of outcome in surgical

sepsis is very likely to aid suitable modification of management strategies [2]. This may improve prognosis in such patients and prevent mortality to some extent. This scoring system also guides the efficient utilization of hospital resources, especially in a resource starved setting. This helps in preventing dumping of valuable drugs and treatment modalities in a patient, who may not survive in spite of all efforts. On the contrary they can be utilized for a person, who may improve well with such costly intervention. Also, the score can be a useful in clinical research tool to evaluate various therapeutic interventions in early sepsis.

AIMS AND OBJECTIVES

1. To assess the incidence and severity of organ dysfunction in patients with sepsis in surgical unit

2. To predict the outcome among them

REVIEW OF LITERATURE

HISTORY

The word sepsis was first coined by Hippocrates (460-370bc) and it is derived from greek word .

Figure 1 Ignaz Semmelweis

Ignaz Semmelweis (1818-1865) was the first researcher who developed on modern aspects of sepsis [(43)]. He was an obstetrician at the Vienna General Hospital, during that time when the death of women in childbed from puerperal fever was a common complication. He found that there was highest mortality in his department of around 18%. Semmelweis discovered that it was common for medical students to examine pregnant women directly after conducting post mortem. Hygenic measures such as hand washing or surgical gloves were not practiced in those times. Semmelweis detected that childbed fever was caused by "decomposed animal matter that entered the blood system"[(4)]. As a matter of

fact, he lowered the mortality rate to 2.55% by introducing hand washing using chlorinated lime solution before any gynaecological procedures.

However, these hygienic measures were not accepted and his colleagues harassed him and made him to leave the city .In 1863, after around 15yrs of his findings, his work was published as "Aetiology, terminus and prophylaxis of puerperal fever" (Die Aetiologie, der Begriff und die Prophylaxis des Kindbettfiebers)[42]. Semmelweis later died from wound infection. It is an irony of fate that he died from the same disease that he detected.

William Osler, was considered as father of American medicine, made his observations in treatise The Evolution of Modern Medicine: "Except on few occasions, the patient appears to die from the body's response to infection rather than from it."[5]

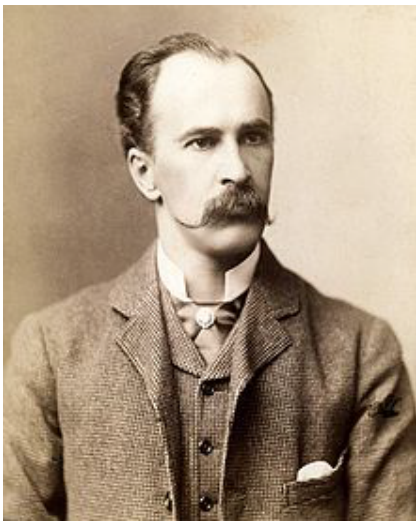

Figure 2: William Osler

From 1878 - 1880, Robert Koch, [41] District Medical Officer for Wollstein. He developed the following four postulates to identify the ssociation between organisms with specific diseases:

1. The microorganism or other pathogen must be present in all cases of the disease.

2. The pathogen can be isolated from the diseased host and grown in pure culture.

3. The pathogen from the pure culture must cause the disease when inoculated into a healthy, susceptible laboratory animal.

4. The pathogen must be re-isolated from the new host and shown to be the same as the originally inoculated pathogen.

Figure 3: Robert Koch

The French chemist Louis Pasteur (1822-1895) discovered that a single cell organisms caused putrefaction. He found that as microbes and these are responsible for causing the disease.[6] He also found that the bacteria can be killed by heating at specific temperature.

This meant that a fluid could be sterilised. He was able to elucidate the principle that contagious diseases are caused by specific microbes and that these microbes are foreign to the infected organism.

DEFINITIONS

In 1992, Bone and colleagues convened a consensus conference on the problem of organ damage caused by excessive activation of the endogenous inflammatory response. They defined four sepsis-related clinical syndromes. These four syndromes were defined in pathophysiologic terms as a hierarchy corresponding to four steps of increasingly exaggerated inflammatory responses- SIRS, sepsis, severe sepsis and septic shock.[40] The first category of SIRS is caused by inflammatory mediators released by lymphocytes, macrophages, granulocytes, and vascular endothelial cells. These activated immune cells release cytokines, enzymes, and oxygen radicals that are beneficial because they can destroy invading microorganisms. These immune mediators also initiate coagulation pathway, amplify the release of additional cytokines and vasoactive agents, and increase capillary membrane permeability.

Infection is defined as presence of microorganism in the body tissue or in blood stream associated with inflammatory response to that organism . At the site of infection the classic findings of rubor, calor, and dolor in areas like the skin or subcutaneous tissue are common.

SIRS Criteria is as follows:

- Heart rate ≥ 90 beats/min
- Respiratory rate ≥20/min OR PaCo2<32mmHg
- Temperature ≥38°C (100.4oF) or ≤36°C (96.8oF
- WBC total count ≥12,000/mL or ≤4,000/mL

SIRS can be caused by a variety of disease processes, including pancreatitis, polytrauma, malignancy, and transfusion reaction, and infection. SIRS caused by infection is termed *sepsis*, and is mediated by the production of a cascade of pro-inflammatory mediators produced by exposure to microbial products.

Septic shock is a medical condition as a result of severe infection and sepsis, though the microbe may be systemic or localized to a particular site. It can cause multiple organ dysfunction syndrome (formerly known as multiple organ failure) and death[(39)]. Its most common victims are children, immuno-compromised individuals, and the elderly, as their immune system cannot deal with infection as effectively as those of healthy adults.

Frequently, patients suffering from septic shock are cared for in intensive care unit. The mortality rate from septic shock is approximately 25–50%.[38] Septic shock is defined as stage of SIRS (Systemic inflammatory response syndrome), in which sepsis, severe sepsis and multi organ dysfunction were considered as different stages of its patho-physiological process.

PATHOPHYSIOLOGY

The pathophysiology of shock in sepsis is multidimensional and complex because of the interaction of multiple physiologic and inflammatory events (Fig 4). The majority of patients with septic shock have hypotension associated with arterial vasodilation. A minority of patients in septic shock are hypovolemic as a result of inflammatory edema or fluid loss and they have a hemodynamic pattern of marked vasoconstriction and low-flow shock. Typically, patients in vasodilatory septic shock have cardiac output 2 fold or greater than normal associated with mean arterial pressure less than 65 mm Hg. The reduction in systemic vascular resistance (SVR)in these patients is attributed to vasodilation in organs with high capillary density, like skin and skeletal muscle.[7]

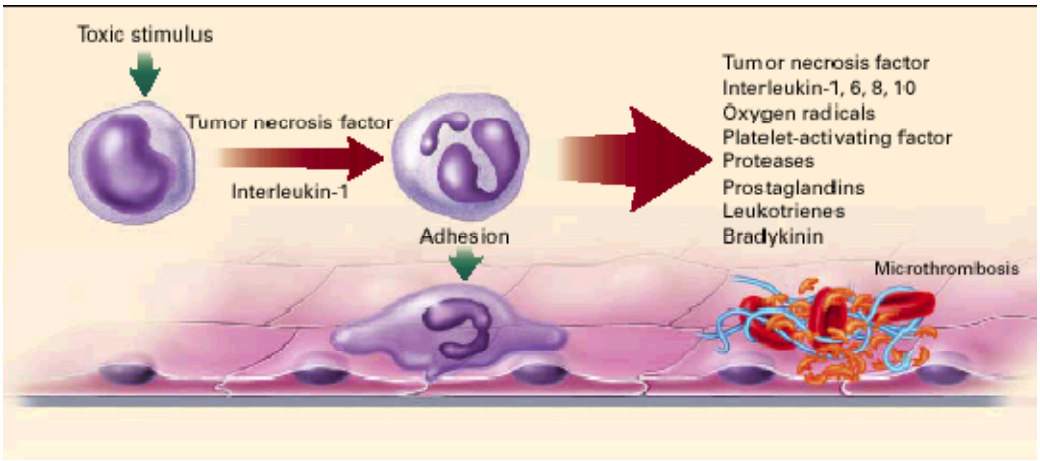

Figure 4: Initiation Of Inflammatory Response

Experimental evidence indicates that excessive production of nitric oxide, a potent vasodilator, is a primary mechanism for the reduced SVR in patients with septic shock. Because of the induction of a potent enzyme system, patients with severe sepsis and septic shock produce large amounts of nitric oxide, and elevated generation of nitric oxide near vascular smooth muscle overwhelm the vasoconstrictive effects of the endogenous vasoconstricting hormones (α-adrenergic catecholamine, angiotensin II, and vasopressin)[(44)]. In clinical trials, treatment of patients in vasodilatory shock with inhibitors of nitric oxide synthesis did not improve their outcome[(37)]. Further research is needed to identify treatments that effectively modify the adverse influence of nitric oxide in septic shock.[(45)]

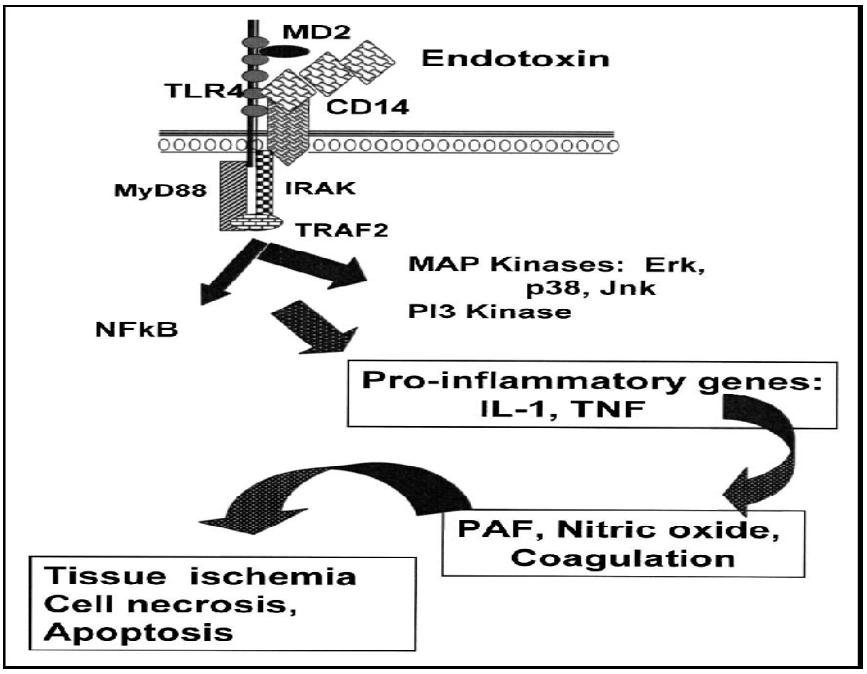

Figure 5 : **Pathway Showing Inflammatory Response**

MULTI ORGAN DYSFUNCTION SYNDROME

The abnormal function or failure of more than one organ or organ system requiring medical support to maintain homeostasis is called MODS[8]. In a susceptible individual, under the influence of associated co-morbidities, the organ systems fail one by one ultimately leading to a complicated disease process and death.

Pathogenesis: The general principles governing the syndrome of multiorgan dysfunction are,[46]

1) Organ failure, no matter how defined, must persist beyond 24 hours
2) Mortality risk increases as the patients accrue additional failing
3) Prognosis is worsened by increased duration of organ failure.

These observations remain true across various critical care settings all over the world. Systemic inflammatory response syndrome (SIRS) is the common basis for multi organ system failure. Infection is by far the commonest cause of SIRS. Though other triggers like pancreatitis, trauma and burns etc can also elicit a similar response.

MANAGEMENT OF SEPTIC SHOCK[33]

Treatment of septic shock consists of following methods

- Volume resuscitation
- Early antibiotic administration
- Early goal directed therapy
- Rapid source identification and control.
- Support of major organ dysfunction.
- Sequestration of lipopolysaccharides.

Treatment guidelines call for the administration of broad-spectrum antibiotics within the first hour following recognition of septic shock. Prompt antibiotics is critically important, as risk of dying increases by approximately 10% for every hour of delay in receiving antibiotics[32]. Time constraints do not allow the culture, identification and testing for antibiotic sensitivity of the specific microorganism responsible for infection. Therefore, combination antimicrobial

therapy, which covers a wide range of potential causative organisms, is tied to better outcomes.

Because lowered blood pressure, in septic shock contributes to poor perfusion, fluid resuscitation is an initial treatment to increase blood volume. Crystalloids such as normal saline and lactated Ringer's solution are recommended as the initial fluid of choice[9], while the use of colloid solutions such as hydroxyethyl starch have not shown any advantage or decrease in mortality. When large quantities of fluids are given, administering albumin has shown some benefit.

Among the choices for vasopressors, norepinephrine is superior to dopamine in septic shock. Norepinephrine is the preferred vasopressor, while epinephrine can be added to norepinephrine when needed. Low dose vasopressin may also be used as an addition to norepinephrine, but is not recommended as a first-line treatment. Dopamine can cause rapid heart rate and arrhythmias, and is only recommended in combination with norepinephrine in those with slow heart rate and low risk of arrhythmia[31]. In the initial treatment of hypotension in septic shock, the goal of vasopressor treatment is a mean arterial pressure (MAP) of 65mm Hg.[47]

Antimediator agents may be of some limited use in severe clinical situations however are controversial:

- Low dose steroids (hydrocortisone) for 5 – 7 days led to improved outcomes.

- Recombinant activated protein C (drotrecogin alpha) in a 2011 concrane review was found not to decrease mortality and thus was not recommended for use. Other reviews however comment that it may be effective in those with very severe disease. The first and only activated protein C drug, drotrecogin alfa (Xigris)[10], was voluntarily withdrawn in October 2011 after it failed to show a benefit in patients with septic shock, including the more severe disease .

Respiratory	SaO_2>90â€“95% (may have to settle for lower) Permissive hypercapnia
Cardiovascular	Maintain cardiac output/oxygen delivery and blood pressure compatible with adequate organ perfusion (e.g. no metabolic acidosis)
Renal	Maintain adequate metabolic and fluid homeostasis by intravascular filling, diuretics, vasoactive agents, and/or haemo(dia)filtration
Haematological	Maintain haemoglobin >7g/dl (unless cardiorespiratory problems), platelets >20â€“40 Ã— 10^9/l, INR <1.5â€“2.5
Gastrointestinal	Stress ulcer prophylaxis (generally by enteral nutrition), pancreatitis, acalculous cholecystitis
Infection	Antibiotics, pus drainage, good infection control
Nutrition	Preferably early and by enteral route
Pressure area/mouth/joint care	Frequent turns, low pressure support surfaces, nursing care and physiotherapy
Psychological	Support to both patient and family

Figure 6 : **Management of Septic Shock**

SCORING SYSTEMS

Typically, scoring systems are used to quantify the severity of illness of a study population, to compare different populations by summarizing the cases, and more recently, as an entry criteria for certain interventional studies. They are also used to compare actual versus expected outcomes for a specific physician, ICU, hospital, or region [30]. Although these systems can be applied to individual patients to predict outcome, that practice is controversial. Previous investigators have identified a link between the number of dysfunctional organs and both short-term and long-term mortality among emergency department patients with infection. A few of the most commonly used such scoring systems are

- APACHE
- SOFA
- Simplified acute physiology score (SAPS)
- Mortality probability model (MPM)
- Therapeutic intervention scoring system (TISS)
- Logistic organ dysfunction score (LODS)
- Multiorgan dysfunction score (MODS)

APACHE SCORING SYSTEM [48]

Knaus et al developed APACHE scoring system in 1985. It consists of 12 physiological variables calculated by multivariate analysis. The scores ranges from 0 – 71. The data of APACHE II are calculated using the equation[11].

In Hospital Mortality .

(R/1-R) = -3.517 + (APACHE □ × 0.146 + S + D)

R = Risk of death in hospital, S = Risk due to emergency surgery, and D = Risk due to any specific disease.

A score of 25 or less denotes less than 50% mortality and score of 35 or more denotes more than 85% mortality. Although APACHE II score provides severity of illness of particular group of patients, they provide little information about the risk of individual patients. As an improvised version of APACHE II, APACHE III and IV were designed for better prediction.[29]

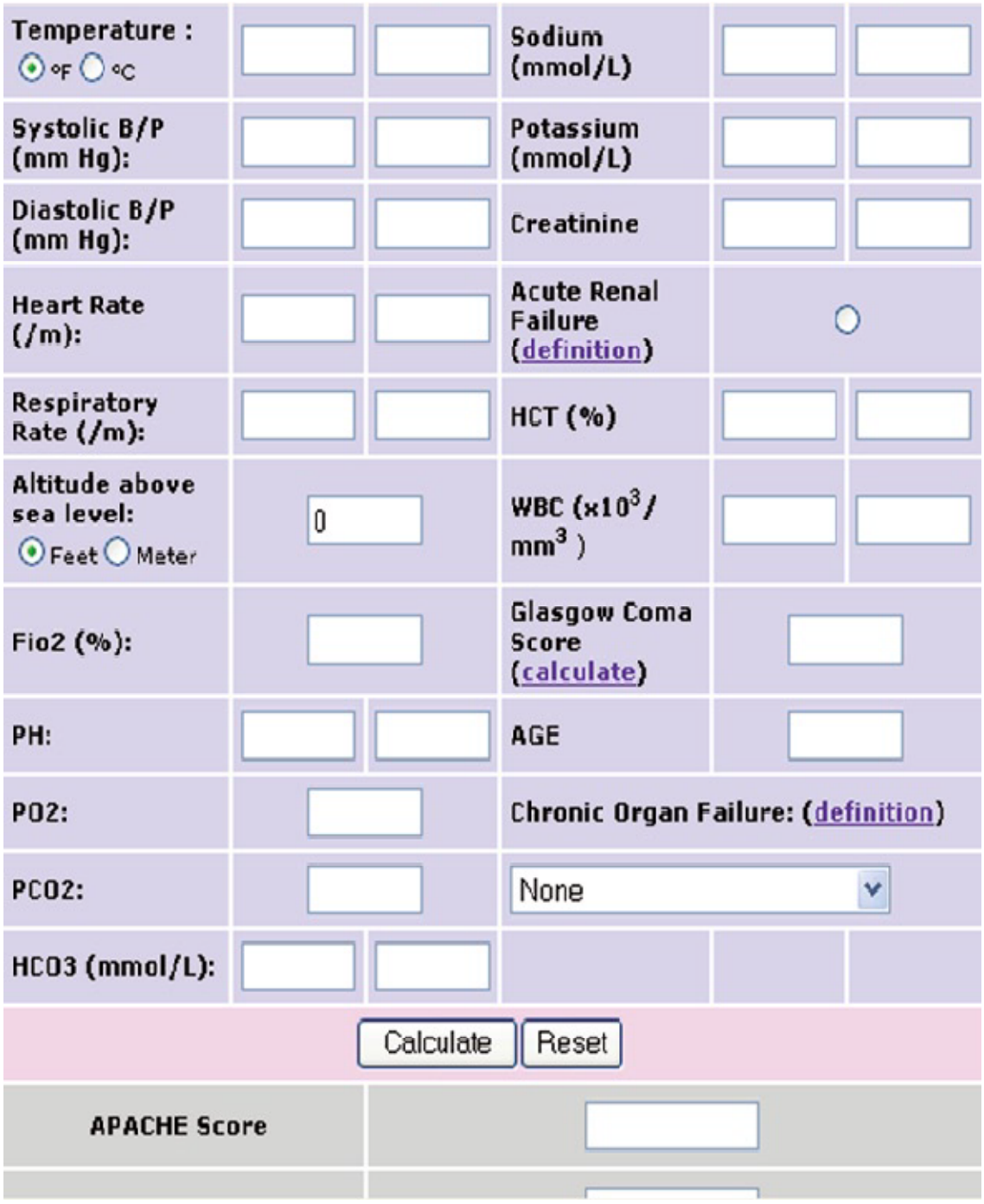

Figure 7 : APACHE Scoring System

SIMPLIFIED ACUTE PHYSIOLOGY SCORE (SAPS)

Simplified acute physiology score was introduced by Le Gall et al in 1984[28]. It was designed to encounter the difficulties faced during assessment of APS used in APACHE score. It was calculated by taking the 13 most easily measurable physiological variables used in APACHE score. The total score is obtained as the highest score of ICU admission in the first 24 hours. SAPS had its advantage over APACHE II in accurately predicting mortality in a stratified group of patients.[12]

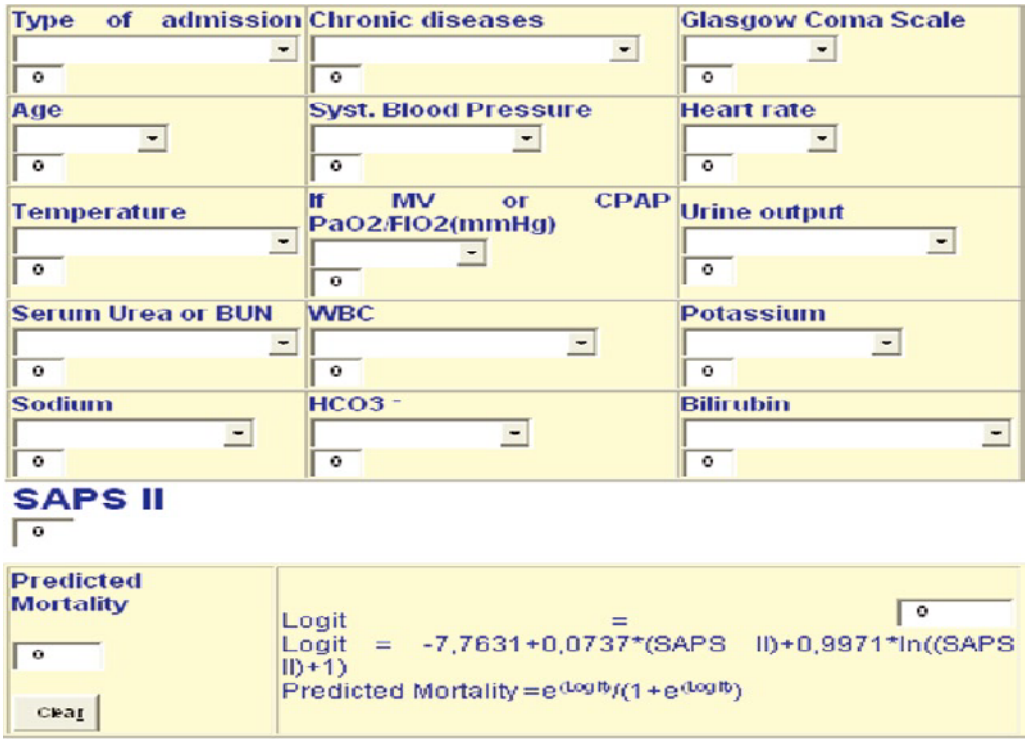

Figure 8 : SAPS Scoring System

MORTALITY PROBABILITY MODEL

Lemeshow et al in 1985, first published the mortality prediction model[49]. He designed 4 models, like probability of death from data collected at ICU admission (MPM_0), Probability of death based on 24 hours data(MPM_{24})probability of death based on 48 hours data(MPM_{48}), probability of death over a period of time based on MPM_0 and change in probability between MPM_0 and MPM_{24} and change in probability between MPM_{24} and MPM_{48}. Lemeshow et al also developed MPM II to assess serial changes in ICU patients over 72 hours of ICU stay(13). Hence this model had a better advantage over APACHE and SAPS since these two models lack ability of serial assessment.

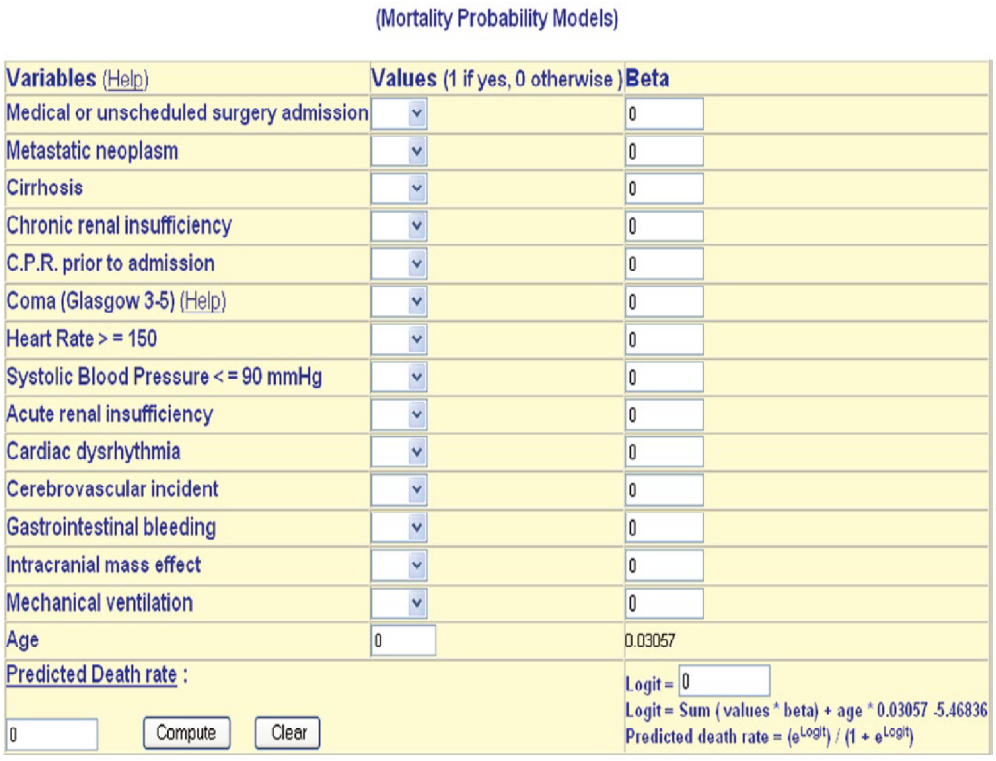

Figure 9: Mortality Probablity Models

THERAPEUTIC INTERVENTION SCORING SYSTEM

Cullen et al in 1974 developed this scoring system[14]. It utilises 76 monitoring and therapeutic parameters. Scores of the first three day ICU stay correlate well with survival. So it is useful in discriminating survivors and non survivors, according to whether the score increases or decreases, respectively.[50]

(Therapeutic Intervention Scoring System - Update 1983)

4 points		3 points	
a. Cardiac arrest and/or countershock within past 48 h	○ yes ○ no	a. Central iv hyperalimentation (includes renal, cardiac, hepatic failure fluid)	○ yes ○ no
b. Controlled ventilation with or without PEEP	○ yes ○ no	b. Pacemaker on standby	○ yes ○ no
c. Controlled ventilation with intermittent or continuous muscle relaxants	○ yes ○ no	c. Chest tubes	○ yes ○ no
d. Balloon tamponade of varices	○ yes ○ no	d. IMV or assisted ventilation	○ yes ○ no
e. Continuous arterial infusion	○ yes ○ no	e. CPAP	○ yes ○ no
f. Pulmonary artery catheter	○ yes ○ no	f. Concentrated K^+ infusion via central catheter	○ yes ○ no
g. Atrial and/or ventricular pacing	○ yes ○ no	g. Nasotracheal or orotracheal intubation	○ yes ○ no
h. Hemodialysis in unstable patient	○ yes ○ no	h. Blind intratracheal suctioning	○ yes ○ no
i. Peritoneal dialysis	○ yes ○ no	i. Complex metabolic balance (frequent intake and output)	○ yes ○ no
j. Induced hypothermia	○ yes ○ no	j. Multiple ABG, bleeding, and/or STAT studies (> 4 shift)	○ yes ○ no
k. Pressure-activated blood infusion	○ yes ○ no	k. Frequent infusion of blood products (>5 units /24 h)	○ yes ○ no
l. G-suit.	○ yes ○ no	l. Bolus iv medication (nonscheduled)	○ yes ○ no
m. Intracranial pressure monitoring	○ yes ○ no	m. Vasoactive drug infusion (1 drug)	○ yes ○ no
n. Platelet transfusion	○ yes ○ no	n. Continuous antiarrythmia infusions	○ yes ○ no
o. IABP (Intra Aortic Balloon Pressure)	○ yes ○ no	o. Cardioversion for arrythmia (not defibrillation).	○ yes ○ no
p. Emergency operative procedures (within past 24 h)	○ yes ○ no	p. Hypothermia blanket	○ yes ○ no
q. Lavage of acute GI bleeding	○ yes ○ no	q. Arterial line	○ yes ○ no
r. Emergency endoscopy or bronchoscopy	○ yes ○ no	r. Acute digitalization - within 48 h	○ yes ○ no
s. Vasoactive drug infusion (> 1 drug)	○ yes ○ no	s. Measurement of cardiac output by any method	○ yes ○ no
		t. Active diuresis for fluid overload or cerebral edema	○ yes ○ no

Figure 10: Therapeutic Intervention Scoring System

SOFA SCORING SYSTEM

The SOFA score was developed in 1994, by the European Society of Intensive Care and Emergency Medicine, to provide a means to describe the degree of organ failure in individuals and groups of ICU patients. Vincent et al published the SOFA score and proved that infected patients had more risk of organ dysfunction than the non- infected .[15]

Table 3 Sequential organ failure assessment score

Organ system	Score				
	0	1	2	3	4
Respiratory PaO_2/FiO_2	> 400	≤ 400	≤ 300	≤ 200	≤ 100
Renal creatinine (μmol/L)	≤ 110	110-170	171-299	300-440 urine output ≤ 500 mL/d	> 440 urine output < 200 mL/d
Hepatic bilirubin (μmol/L)	≤ 20	20-32	33-101	102-204	> 240
Cardiovascular hypotension	No hypotension	MAP < 70 mmHg	Dopamine ≤ 5[1] Dobutamine (any dose)	Dopamine > 5[1] or epinephrine ≤ 0.1[1] or norepinephrine ≤ 0.1[1]	Dopamine > 15[1] or epinephrine > 0.1[1] or norepinephrine > 0.1[1]
Hematologic platelet count (/mL)	> 150	≤ 150	≤ 100	≤ 50	≤ 20
Neurologic Glasgow coma score	15	13-14	10-12	6-9	< 6

Figure 11 : SOFA Scoring System

SOFA scoring system analyses 6 variables namely [16]

- Pao2/Fio2 ratio(for respiration)
- Platelets(for coagulation)
- Bilirubin (for liver function)
- Creatinine (for renal function)
- Glasgow coma scale(to assess level of consciousness)
- Blood pressure and the need for inotropic support.

A score of 0 to 4 is given for each of these six variables and a score is obtained using sum total value of each of these parameters. The worst values on each day are recorded and organ function total score can thus be monitored over time.

The increasing SOFA score and the mean SOFA score are highly useful in assessing prognosis and risk stratification of patients.

PARAMETERS

PAO2/FIO2 RATIO:

It is simply defined as the amount of inspired oxygen that reaches the blood. It is impaired in case of lung injury due to any cause. It is also called

carrico index[27]. According to AECC criteria, acute respiratory distress syndrome is diagnosed , if Pao2/Fio2 ratio is less than or equal to 200.[17]

Pao2 is the partial pressure of oxygen in the arterial blood. It is measured in millimetres of mercury (mmHg) or torr units. It is measured by an arterial blood gas analyser(ABG). Normal Pao2 is 75 – 100mmHg.

Fio2 is the percentage of oxygen in the inspired mixture of air. Normal Fio2 in inspired atmospheric air is 0.21(21%). In a mechanical ventilator it is usually set as 30 – 40%. In a mechanically ventilated patient 100% oxygen is not administered due to high risk of oxygen toxicity.

Kerbing and his co workers assessed the clinical relevance of variation in Pao2/Fio2 ratio. They demonstrated the clinical utility of this parameter.

The Pao2/Fio2 scores are

- Score 0 – more than 400
- Score 1 – less than or equal to 400
- Score 2 – less than or equal to 300
- Score 3 – less than or equal to 200
- Score 4 – less than or equal to 100.

Figure 12 : Arterial blood gas analyser (ABG)

CREATININE

In SOFA scoring serum creatinine values are estimated periodically to assess the renal function over a period of time till the patient is in icu. Creatinine is a breakdown product of creatine phosphate, which is found in muscle. Each day 1-2 % of muscle creatine is converted to creatinine. It is excreted both by glomerular filtration and tubular secretion. Rise in serum creatinine is a marker of damage to nephrons. Normal serum values are 0.7 – 1.2(males) and 0.5 – 1.0(females) [18]. Impaired renal function can be due to pre renal, renal or post renal causes. Some of the commonest causes of renal failure are

- Severe dehydration
- Acute pyelonephritis
- Diabetes
- Hypertension
- Renal calculi
- Hemorrhagic fevers
- Disseminated intravascular coagulation
- Autoimmune and other connective tissue disorders.

The scores used for creatinine in SOFA score are,[19]

- Score 0 – less than 1.2 mg/dl
- Score 1 – 1.2 to 1.9 mg/dl
- Score 2 – 2.0 to 3.4 mg/dl
- Score 3 – 3.5 to 4.9 mg/dl
- Score 4 – more than 5 mg/dl

PLATELET COUNT

Platelet count is used as a parameter in SOFA score to assess coagulation function and its impairment during disease states. The coagulation mechanism involves activation, adhesion and aggregation of platelets in response to a stimuli, say an injury or infection. Both platelet number and function should be adequate for this function to be intact. Coagulation cascade is one of the best understood system in humans. Primary hemostasis is mainly due to platelets, which is characterised by formation of platelet plugs. Activated platelets release stored granules into the blood. These granules contain

- Serotonin
- ADP
- Platelet activating factor
- Platelet factor 4
- Vonwillebrand factor
- Thromboxane A_2

All these substances when released into the blood stream activate additional platelets. These steps lead on to the activation of various enzymes of coagulation cascade resulting in activation of clotting factors, which is called secondary hemostasis. Various systemic illness can be associated with a

decreased platelet count. It can be either due to decreased production, increased destruction or impairment of platelet function.

Causes of thrombocytopenia [20]

- Vitamin B12 and folate deficiencies
- Infections like HIV disease
- Leukemias
- Disseminated intravascular coagulation
- Thrombotic thrombocytopenic purpura
- Viral infections
- Gram negative septicaemia
- Heparin induced thrombocytopenia
- Radiation induced bone marrow suppression
- Drug toxicity

The scores used for platelet count in SOFA are

- Score 0 - >150 × 10^3/mm^3
- Score 1 - <150× 10^3/mm^3
- Score 2 - <100× 10^3/mm^3

- Score 3 - <50× 10^3/mm^3
- Score 4 - <20× 10^3/mm^3

BILIRUBIN

` Bilirubin levels are measured as a marker of liver function. Liver plays a pivotal role in regulating a large number of metabolic pathways in the body. Bile is secreted in the hepatic lobules and it drains ultimately into the bile duct after traversing through canaliculi, small bile ducts and larger bile ducts.

It consists of bile acids, phospholipids and unesterified cholesterol. Daily bile output from the liver is 500 – 600ml. It consists of two fractions. Direct or hydrophilic type and indirect or hydrophobic type. Conjugation of indirect to direct fraction takes place in the liver, which is an enzyme mediated process. This whole array of steps in the formation to elimination of bile can be disturbed in disease states. Elevations in bilirubin levels can be used to assess liver function over time[21], which helps in predicting worsening or improvement of liver function in a patient with sepsis.

Some of the conditions in which bilirubin levels are raised are,

- ✓ Acute hepatitis
- ✓ Alcoholic liver disease
- ✓ DIC and septicaemia
- ✓ Hepatocellular carcinoma

- ✓ Haemolytic jaundice
- ✓ Obstructive jaundice
- ✓ Congenital liver enzyme abnormalities
- ✓ Massive blood transfusion

Most biologic system in the body gets affected by excess bilirubin in blood. Normal bilirubin levels in blood are 1.0 to 1.5mg/dl. Upto 30% of that is direct or conjugated bilirubin, which equals 0.3 mg/dl. It is water soluble. The rest of the fraction is insoluble in water and it is called unconjugated bilirubin. This is the toxic form of bilirubin, which when accumulates in excess gets deposited in the brain especially in the basal ganglia which may lead to seizures or neurological deficits.

The scores used for bilirubin are

- ✓ Score 0 - < 1.2 mg/dl
- ✓ Score 1 – 1.2 to 1.9 mg/dl
- ✓ Score 2 – 2.0 to 5.9 mg/dl
- ✓ Score 3 – 6.0 to 11.9 mg/dl
- ✓ Score 4 - >12 mg/dl

GLASGOW COMA SCALE

It gives a reliable and objective way of recording the conscious state of a person. It is easy to use both for the medical and paramedical personnel for initial as well as continuing medical assessment in an ICU(22). It has value in predicting ultimate outcome. Three types of responses are assessed.

GCS scale was used initially only for head injury patients. Now it is being used both for acute medical and trauma patients. It is also being used to monitor patients in ICU in a seriously ill state. The scale was published in 1974 by Graham Teasdale and Bryan J. Jennett, at the University of Glasgow Institute Of Neurological Sciences. Both of them were neurosurgeons.[23]

Glasgow Coma Scale		
Eye Response	Open Spontaneously	4
	Open to Verbal command	3
	Open in response to pain	2
	No response	1
Verbal Response	Talking / Orientated	5
	Confused speech / Disorientated	4
	Inappropriate Words	3
	Incomprehensible sounds	2
	No response	1
Motor Response	Obeys commands	6
	Localizes pain	5
	Withdraws from pain	4
	Abnormal flexion	3
	Extension	2
	No response	1

Figure 13 : Glasgow Coma Scale

The highest possible score is 15, that is in a fully awake person. The lowest possible score is 3, which means deep coma or death.

The scores used for GCS in SOFA are

- ✓ Score 0 – 15
- ✓ Score 1 – 13 to 14
- ✓ Score 2 – 10 to 12
- ✓ Score 3 – 6 to 9
- ✓ Score 4 - <6

BLOOD PRESSURE

"There is no doubt that proper functioning of our pipes and pumps does have an immediate urgency well beyond that of almost any of our other bits and pieces". [(24)]

Steven vogel (vital circuits, 1992)

Hypotension and shock may occur as a final consequence of any organ dysfunction. Maintaining an adequate blood pressure is essential for perfusion and oxygenation of vital organs. In short, shock is a clinical syndrome resulting from inadequate tissue perfusion of any cause, resulting in an imbalance between the requirement and supply of oxygen, causing cellular dysfunction.

This goes on and on like a vicious cycle resulting in cellular death and multi organ dysfunction.

Classification of shock [25]

Hypovolemic	Septic
Traumatic	Hyperdynamic(early)
Cardiogenic	Hypodynamic(late)
Intrinsic	Neurogenic
Compressive	Hypoadrenal

Table 1 : Classification of shock

The scores used for blood pressure in SOFA are

- ✓ Score 0 – No hypotension
- ✓ Score 1- Mean arterial pressure <70
- ✓ Score 2 – dopamine infusion </= 5 or requiring dobutamine
- ✓ Score 3 – dopamine infusion >/= 5 or requiring nor epinephrine </=0.1
- ✓ Score 4 – dopamine infusion > 15 or or requiring nor epinephrine 0.1

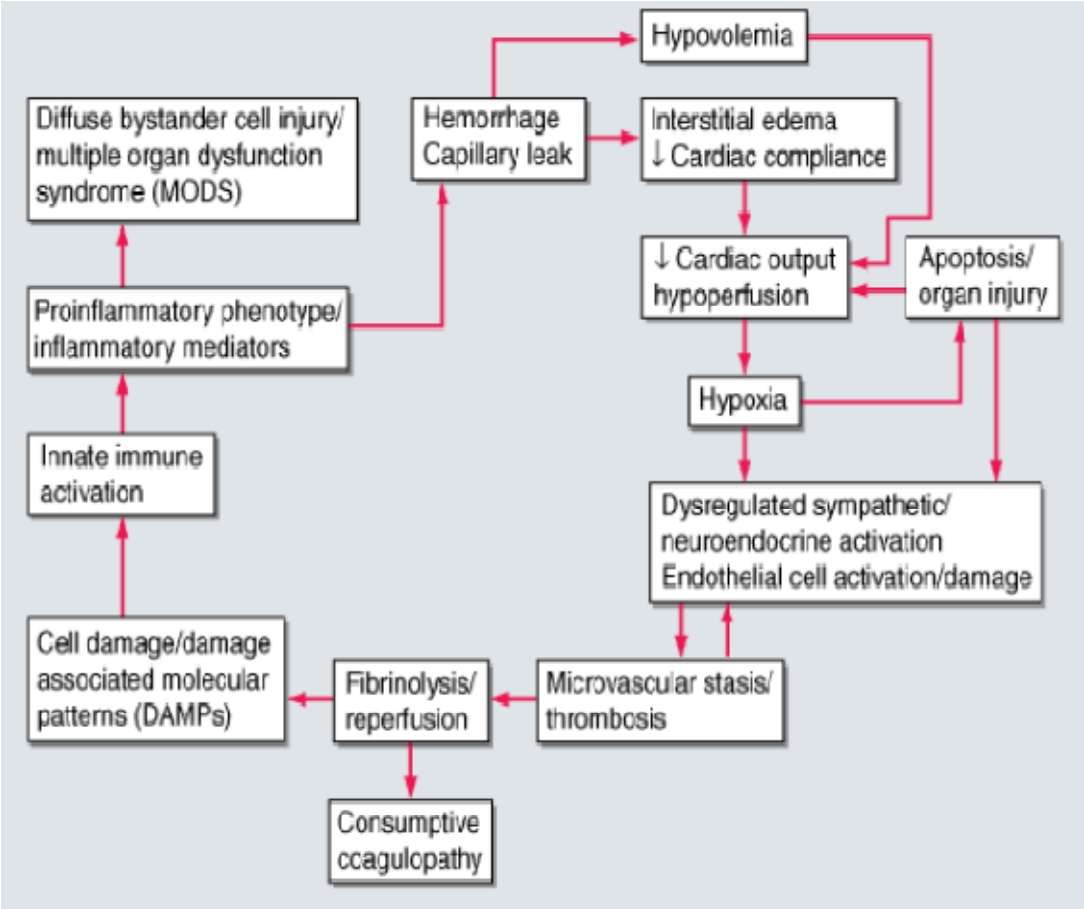

Figure 14 : Shock induced vicious cycle

MATERIALS AND METHODS

100 patients admitted to the surgical unit in Coimbatore medical college hospital with suspected/confirmed sepsis

STUDY DESIGN

Prospective observational study

STUDY GROUP

Patients admitted to the surgical unit at Coimbatore medical college

STUDY DURATION

One year (September 2014- September 2015)

INCLUSION CRITERIA

All patients admitted to the surgical ward with suspected infection, satisfying Two or more criteria of systemic inflammation like

- Heart rate ≥ 90 beats/min
- Respiratory rate ≥20/min OR PaCo2<32mmHg
- Temperature ≥38°C (100.4oF) or ≤36°C (96.8oF
- WBC total count ≥12,000/mL or ≤4,000/mL

Patients which are included in the study are perforation peritonitis with Septicemia, Diabetic ulcer foot with gangrene, Necrotizing fascitis of limbs and abdomen, Burns, Mesenteric ischemia with bowel gangrene, Intestinal Obstruction, Carcinoma, Blunt injury abdomen with solid organ injury.

EXCLUSION CRITERIA

- All patients with age less than 12 years
- All patients who will not give consent for study
- Patients with HIV and chronic renal failure
- Moribund and terminally ill patients with impending mortality within 48-72 hours.

SAMPLE SIZE

A total of 100 patients admitted to Coimbatore medical college surgical unit were studied

CONSENT

Informed consent was taken as per the standard procedures in the institution

ETHICAL CLEARANCE

Obtained from the ethical committee of the institution

PROCEDURE

All patients with suspected/confirmed sepsis admitted in the surgical unit were included in the study. This included operated, non-operated and trauma patients (eg: perforation peritonitis, Diabetic ulcer foot with gangrene Necrotizing fascitis). Patients had to fulfill two or more criteria of systemic inflammation. The parameters involved in calculating the SOFA score were collected on a daily basis . The score was calculated till discharge from ICU, mortality or day7 of admission to ward whichever was the earliest. The SOFA at admission was labelled T0 and at day 2 was labelled as T48(i.e. at 48 hours) and at day 4 was labelled as T96(i.e. at 96 hours) . The difference calculated as Delta SOFA. The Maximum, Mean and total SOFA were also calculated and Compared with outcome of the patient.

Blood Investigations were taken under aseptic conditions with adequate care and sent to the hospital 24 hours laboratory immediately. All the investigations were done in our hospital and no investigations or procedure done outside the hospital. Any experimental or so far unused materials or methods were not used on the patients. Serum bilirubin was calculated using an auto analyser using the method of malloy and evelyn.

ABG was done using ion selective electrode in an ABG analyser. Platelet count was done using sysmex KX21.3 which is an automated cell count analyser, in clinical pathology lab.

STATISTICAL METHODS

This is a prospective non interventional study. Data analysed using SPSS Software Version17. Descriptive statistics are reported using mean, median and SD for continuous variables, number and percentages for categorical variables. Logistic regression was used to find the predictors for mortality. Probability value less than 0.05 was considered statistically significant.

RESULTS

SURVIVORS AND NON SURVIVORS

Among the 100 patients involved in the study 53% survived and 47% succumbed to their illness. The minimum age of the person enrolled in the study was 17 and the maximum age was 85.

SOFA SCORE ON ADMISSION

SOFA score	Survivors	Non survivors	Total
6 – 7	5	1	6
8 – 9	19	7	26
10 – 11	13	4	17
12 and above	16	35	51
Total	53	47	100

Table 2 : SOFA score on admission

The minimum SOFA score of the patients admitted was 6. Hence the data column starts with values of 6 and above. This table shows that there is a sharp rise in non survivors at a SOFA score above 12.

AREA UNDER THE CURVE

Test Result Variable(s):SOFA ADMISSION

Area	Std. Error[a]	Asymptotic Sig.[b]	Asymptotic 95% Confidence Interval	
			Lower Bound	Upper Bound
.760	.048	.000	.665	.855

The test result variable(s): SOFAADMISSION has at least one tie between the positive actual state group and the negative actual state group. Statistics may be biased.

a. Under the nonparametric assumption

b. Null hypothesis: true area = 0.5

ROC CURVE FOR ADMISSION SOFA

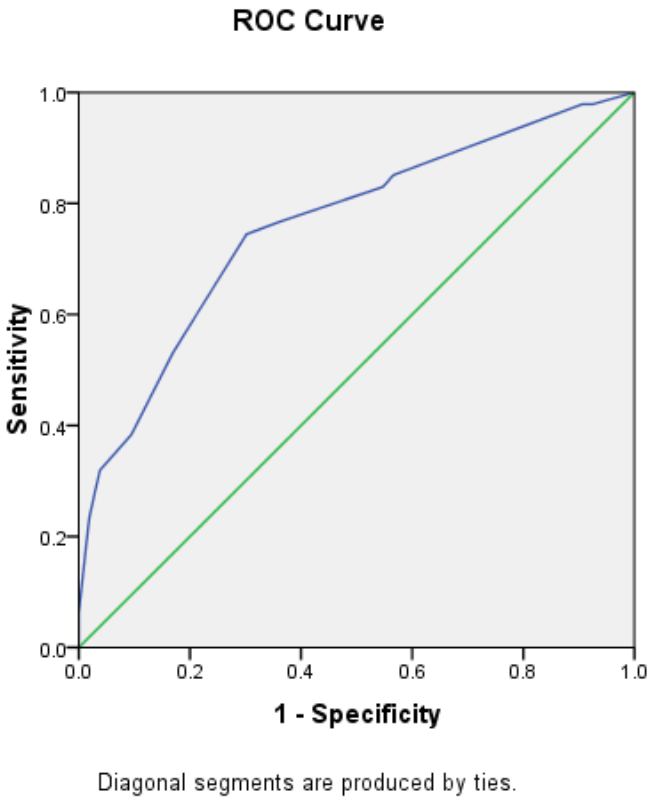

Figure 15 : ROC Curve for Admission Sofa

BAR CHART

NO. OF DEATHS

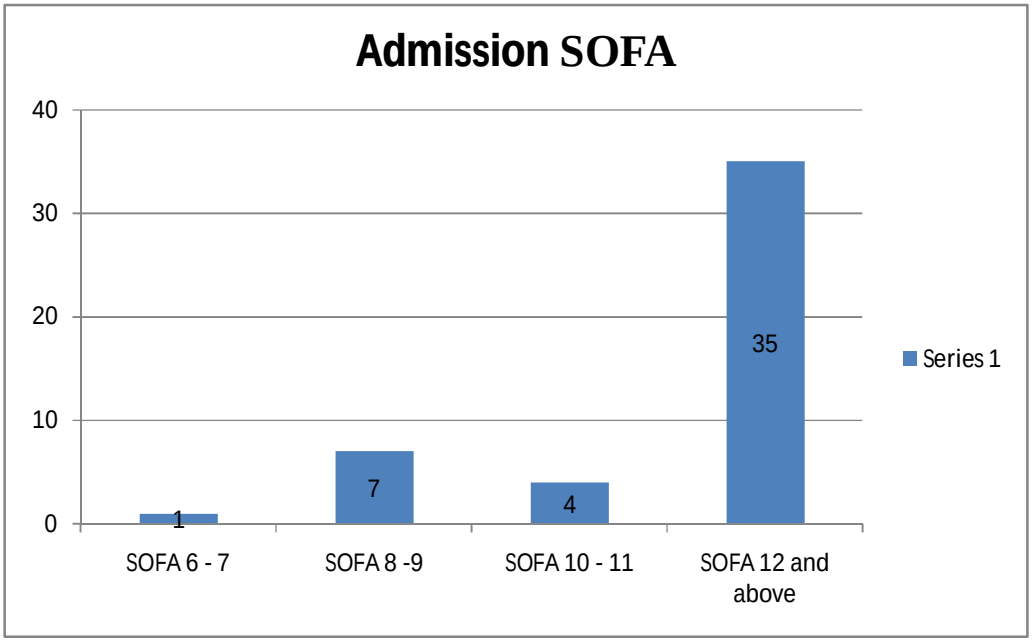

Figure 16: Comparision between Admission SOFA and No. of Deaths

The minimum admission SOFA score of patients in this study is 6. Among the 6 patients who had this score 1 patient expired. That is, the mortality rate is 16.7 %. Among the 51 patients who had an admission SOFA score of 12 and above 35 patients expired escalating the mortality rate to 68.6%.

SOFA AT 48 HOURS FOR NON SURVIVORS

SOFA SCORE	NO.OF NON SURVIVORS
8 – 9	3
10 - 11	4
12 and above	40

Table 3 : SOFA at 48 hours for non survivor

At 48 hours the minimum SOFA score observed among the study population is 8. Hence the data column starts with 8 and above.

AREA UNDER THE CURVE

Test Result

Variable(s): SOFA48Hr

Area	Std. Error[a]	Asymptotic Sig.[b]	Asymptotic 95% Confidence Interval	
			Lower Bound	Upper Bound
.914	.028	.000	.859	.970

The test result variable(s): SOFA 48Hr has at least one tie between the positive actual state group and the negative actual state group. Statistics may be biased.

a. Under the nonparametric assumption

b. Null hypothesis: true area = 0.5

ROC CURVE FOR SOFA AT 48 HOURS

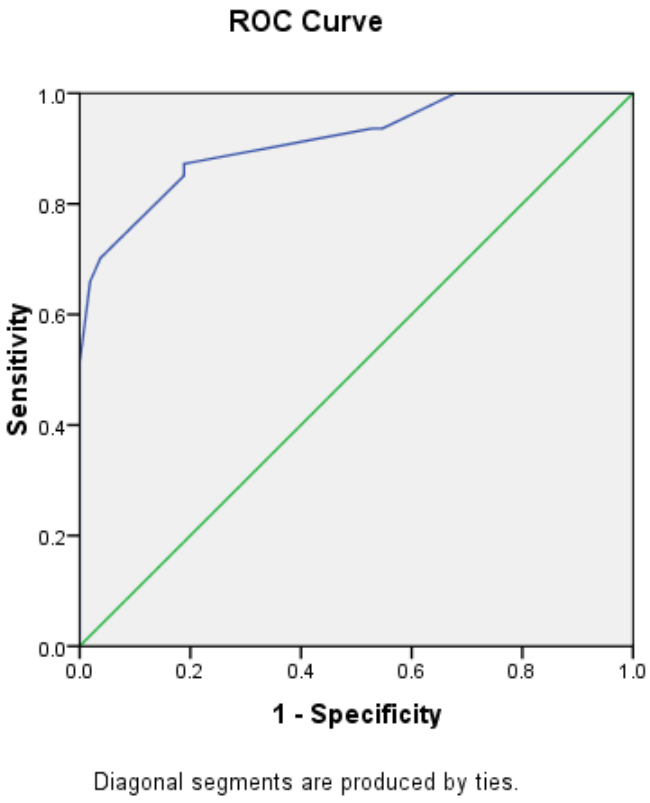

Figure 17: ROC Curve for SOFA at 48 Hours

BAR CHART

NO. OF DEATHS

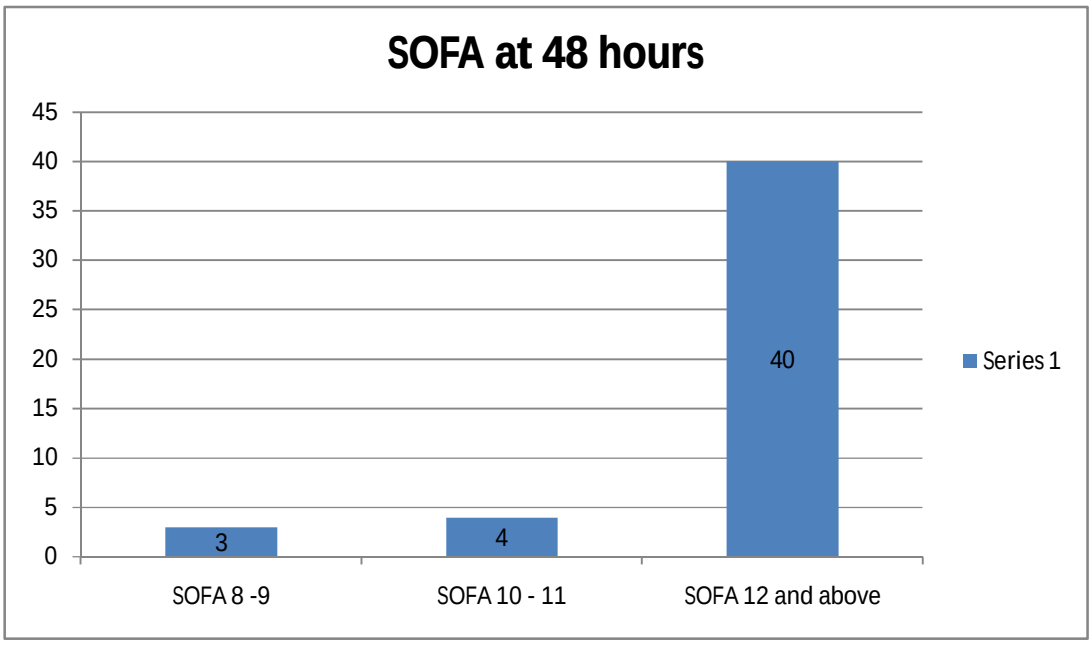

Figure 18: Comparison between SOFA at 48 Hrs and No of Deaths

This picture shows that a SOFA score of 12 and above at 48 hours of admission shows an increase in the number of non survivors. The minimum SOFA score of the study population at 48 hours is 8. Among the 47 non survivors, 3 patients had these minimum score. Patients who had a score of 12 and above were 40.

SOFA SCORE AT 96 HOURS FOR NON SURVIVORS

SOFA SCORE	NO. OF NON SURVIVORS
8 – 9	3
10 – 11	3
12 and above	41

Table 4 : SOFA Score at 96 hours for Non Survivors

AREA UNDER THE CURVE

Test Result Variable(s): SOFA 96HR

Area	Std. Error[a]	Asymptotic Sig.[b]	Asymptotic 95% Confidence Interval	
			Lower Bound	Upper Bound
.937	.023	.000	.892	.982

ROC CURVE AT 96 HOURS

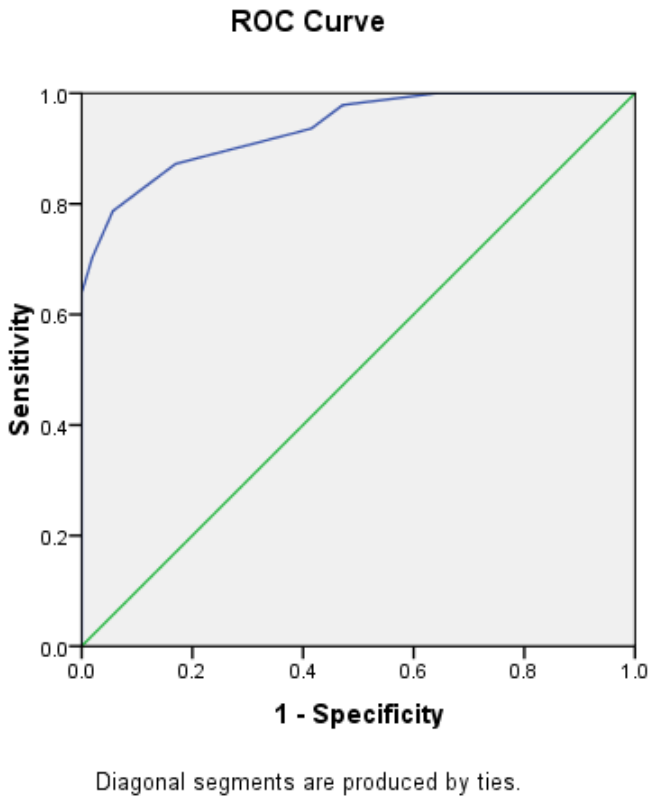

Figure 19: ROC Curve at 96 Hours

BAR DIAGRAM

NO OF DEATHS

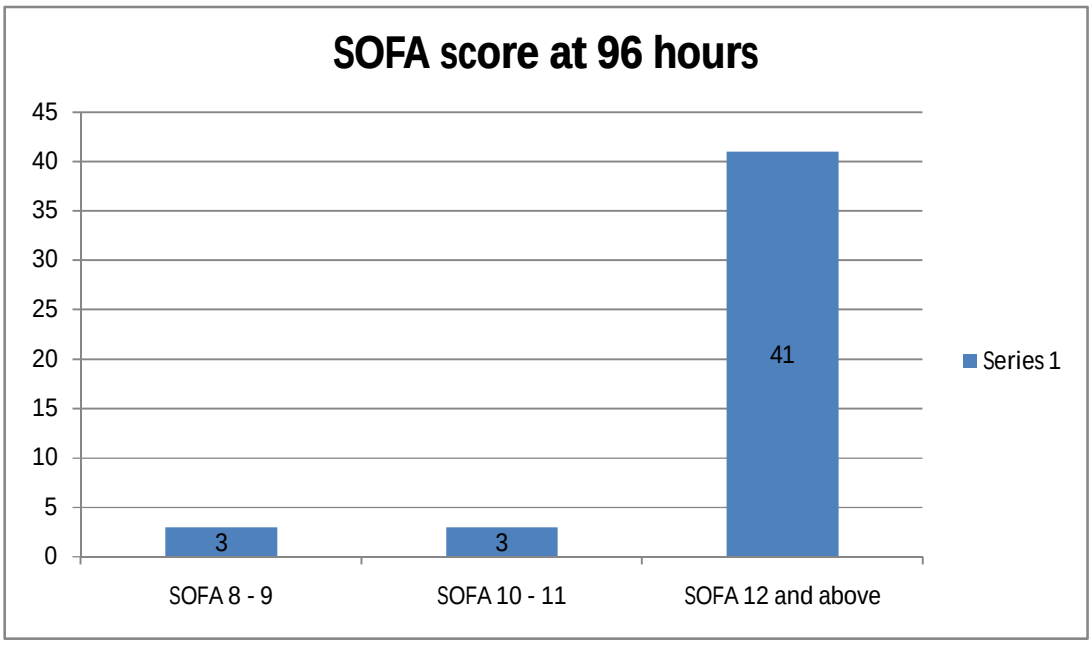

Figure 20: Comparison between SOFA at 96 Hrs and No of Deaths

This chart depicts that survival rate is reduced when the SOFA score increases above 12, at 96 hours of admission. At 96 hours 41 out of the 47 patients expired, had a score of 12 and above.

DELTA SOFA

It is the difference between the subsequent SOFA scores. Δ SOFA 48 is the difference between admission score and the score at 48 hours. ΔSOFA 96 is the difference between the score at admission and 96 hours.

SOFA SCORE 48 HOUR CHANGES

The patient data is analysed as those who decreased, unchanged and increased from the initial score respectively, and the outcome is analysed.

Δ SOFA 48	**Survivors**	**Non survivors**
Decreased	35	6
Unchanged	8	9
Increased	10	32

Table 5 : SOFA score 48 hour changes

AREA UNDER THE CURVE

Test Result Variable(s): SOFA 48 difference

Area	Std. Error[a]	Asymptotic Sig.[b]	Asymptotic 95% Confidence Interval	
			Lower Bound	Upper Bound
.830	.041	.000	.749	.910

SOFA 48 HOUR CHANGES:

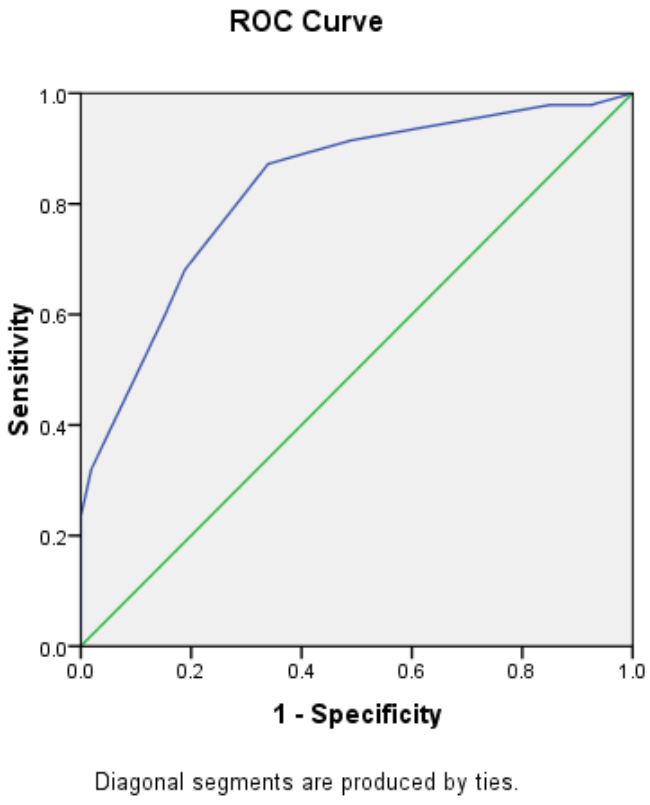

Figure 21 : ROC Curve for SOFA 48 Hrs changes

BAR CHART

NO. OF DEATHS

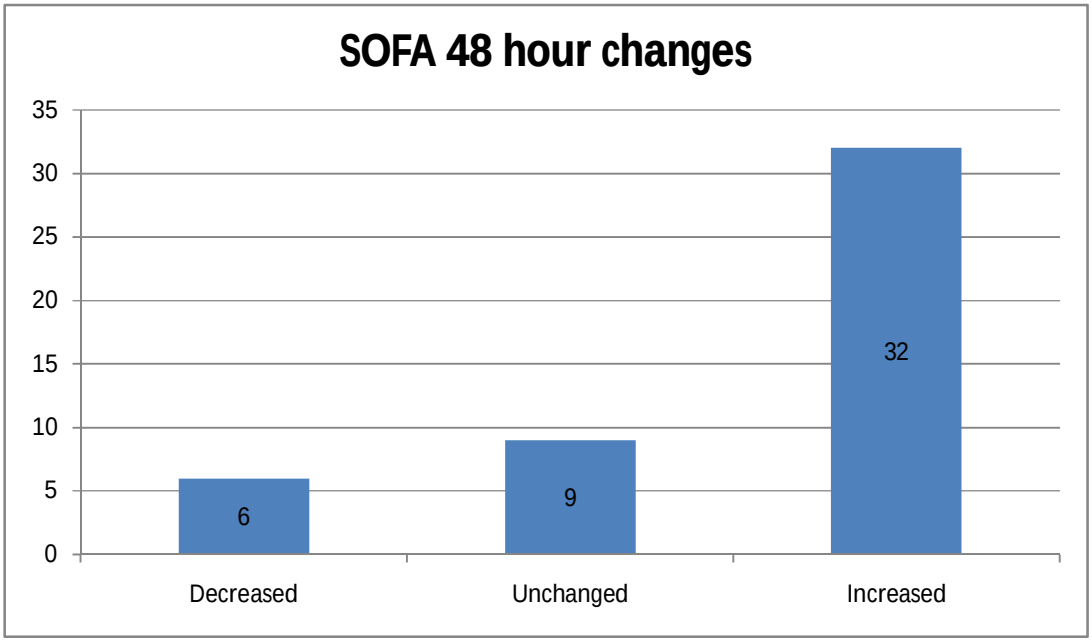

Figure 22: Comparison between SOFA 48 Hrs changes and no of deaths

These data depicts that when the SOFA score is increased from admission to 48 hours, there is an increase in mortality. On contrary the mortality rate has decreased when the score falls. Among the 47 non survivors 32 (68.08%) had an increase in their Δ48 scores.

SOFA SCORE 96 HOUR CHANGES

The patient data is analysed as those who decreased, unchanged and increased from the initial score respectively, and the outcome is analysed.

Δ SOFA 96	**Survivors**	**Non survivors**
Decreased	39	7
Unchanged	7	2
Increased	7	38

Table 6 : SOFA score 96 hour changes

SOFA 96 HOUR CHANGES

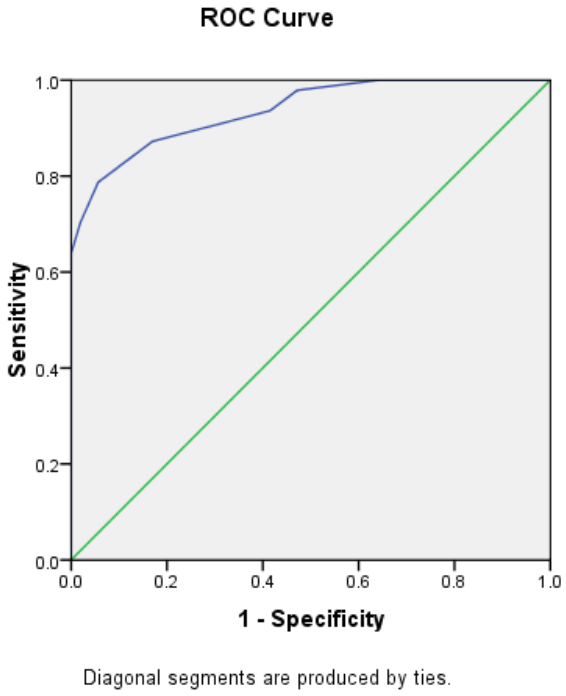

Figure 23 : ROC Curve of SOFA 96 Hrs Changes

BAR CHART

NO. OF DEATHS

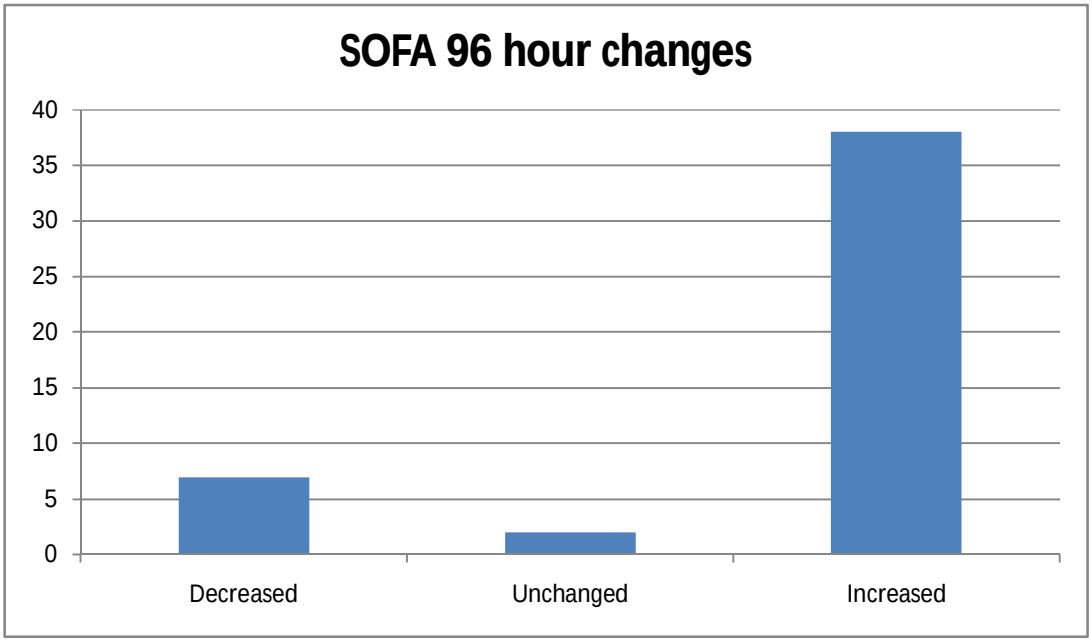

Figure 24: Comparison between SOFA 96 Hrs change and no of deaths

This chart depicts mortality rate is increased when the SOFA score is increased from admission to 96 hours. On contrary, the mortality rate has decreased when the score falls. Among the 47 non survivors 38 (80.85%) had an increase in their Δ 96 scores.

MEAN SOFA

Mean SOFA calculates the average value of the prognostic score during the entire hospital stay of the patient.

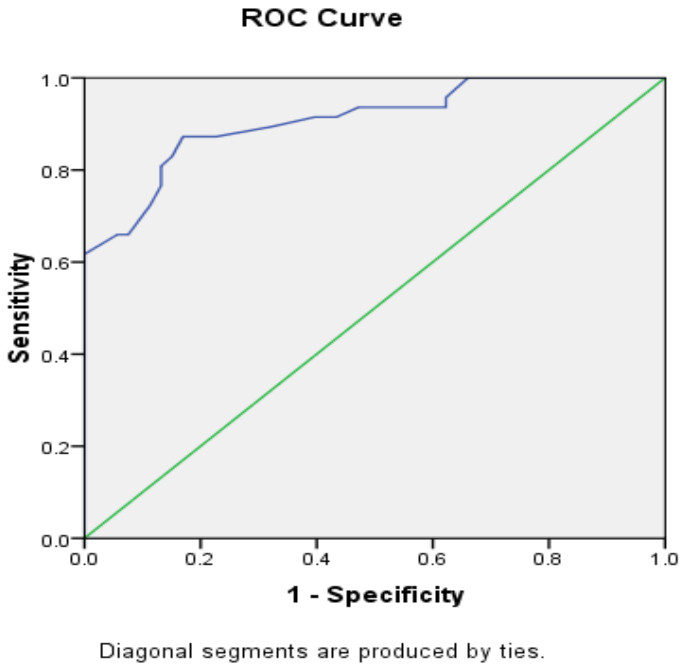

Figure 25: ROC Curve for Mean SOFA

AREA UNDER THE CURVE

Test Result Variable(s): MEAN SOFA

Area	Std. Error[a]	Asymptotic Sig.[b]	Asymptotic 95% Confidence Interval	
			Lower Bound	Upper Bound
.908	.029	.000	.851	.966

COORDINATES OF THE CURVE

Test Result Variable(s): MEAN SOFA

Positive if Greater Than or Equal To[a]	**Sensitivity**	**1 – Specificity**
4.3333	1.000	1.000
5.6667	1.000	.962
6.3333	1.000	.925
7.0000	1.000	.755
7.5000	1.000	.660
7.8333	.979	.642
8.1667	.957	.623
8.5000	.936	.623
8.8333	.936	.491
9.1667	.936	.472
9.5000	.915	.434
10.0000	.915	.396

10.5000	.894	.321
10.8333	.872	.226
11.167	**0.87**	**0.17**
11.5000	.830	.151
11.8333	.809	.132
12.1667	.766	.132
12.5000	.723	.113
12.8333	.660	.075
13.1667	.660	.057
13.5000	.617	.000
13.8333	.574	.000
14.1667	.532	.000
14.5000	.489	.000
14.8333	.404	.000
15.1667	.383	.000
15.5000	.319	.000
16.1667	.277	.000
16.8333	.213	.000
17.1667	.191	.000
17.5000	.149	.000
18.0000	.106	.000
18.5000	.064	.000
20.0000	.021	.000
22.3333	.000	.000

The test result variable(s): MEAN SOFA has at least one tie between the positive actual state group and the negative actual state group.

The smallest cutoff value is the minimum observed test value minus 1, and the largest cutoff value is the maximum observed test value plus 1. All the other cutoff values are the averages of two consecutive ordered observed test values. These data shows that, a mean SOFA score of 11 and above is an excellent predictor of mortality, above which the number of non survivors increase.

TOTAL SOFA

It is the sum total of all the scores obtained from an individual patient during his hospital stay. It gives information about the severity of the illness since it gives the total worst score of all organs.

AREA UNDER THE CURVE

Test Result Variable(s):TOTALSOFA

Area	Std. Error[a]	Asymptotic Sig.[b]	Asymptotic 95% Confidence Interval	
			Lower Bound	Upper Bound
.908	.029	.000	.851	.966

COORDINATES OF THE CURVE

Test Result Variable(s): TOTAL SOFA

Positive if Greater Than or Equal To[a]	Sensitivity	1 – Specificity
15.0000	1.000	1.000
17.0000	1.000	.962
19.0000	1.000	.925
21.0000	1.000	.755
22.5000	1.000	.660
23.5000	.979	.642
24.5000	.957	.623
25.5000	.936	.623
26.5000	.936	.491
27.5000	.936	.472
28.5000	.915	.434
30.0000	.915	.396
31.5000	.894	.321
32.5000	.872	.226
33.500	**0.87**	**0.17**
34.5000	.830	.151
35.5000	.809	.132
36.5000	.766	.132
37.5000	.723	.113
38.5000	.660	.075
39.5000	.660	.057

40.5000	.617	.000
41.5000	.574	.000
42.5000	.532	.000
43.5000	.489	.000
44.5000	.404	.000
45.5000	.383	.000
46.5000	.319	.000
48.5000	.277	.000
50.5000	.213	.000
51.5000	.191	.000
52.5000	.149	.000
54.0000	.106	.000
55.5000	.064	.000
60.0000	.021	.000
65.0000	.000	.000

The test result variable(s): TOTAL SOFA has at least one tie between the positive actual state group and the negative actual state group.

The smallest cutoff value is the minimum observed test value minus 1, and the largest cutoff value is the maximum observed test value plus 1. All the other cutoff values are the averages of two consecutive ordered observed test values

These data depict that a total SOFA score of 33 and above is an excellent predictor of mortality, above which the number of non survivors increase.

OUTCOME BASED ON SEX

Sex	Survivors	Non survivors	Total
Male	33	36	69
Female	20	11	31
Total	53	47	100

Table 7 : Outcome Based on Sex

GRAPHIC REPRESENTATION

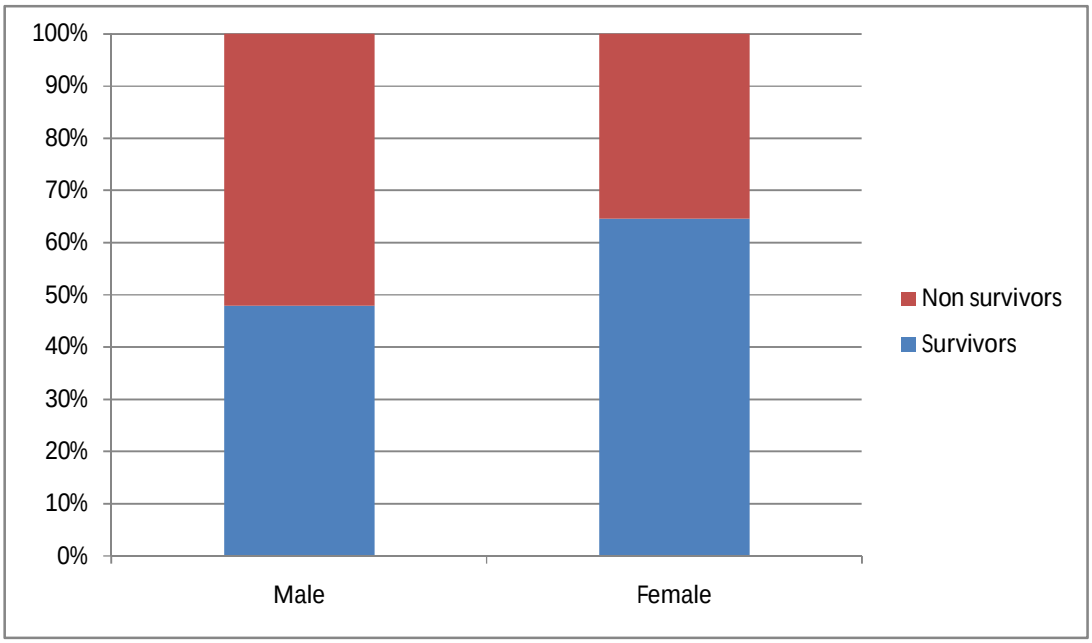

Figure 26: Comparison between outcome and Sex

Out of 69 male patients, 36 (52.2%) patients expired and out of 31 female patients, 11(35.5%) patients expired.

OUTCOME FOR VENTILATOR SUPPORT

Mechanical Ventilation status	Survivors	Non survivors
Ventilated	8	33
Non ventilated	45	14

Table 8 : Outcome for ventilator support

GRAPHIC REPRESENTATION

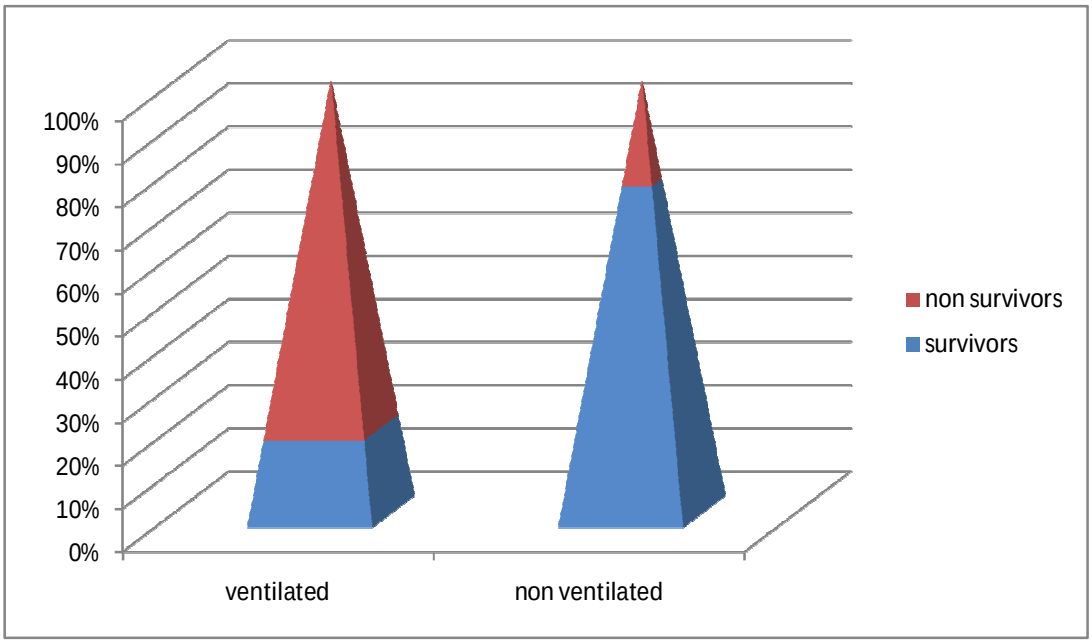

Figure 27: Comparison between Outcome and Ventilator Support

Among the 41 patients ventilated 33 (80.5%) expired and among the 59 patients who did not require ventilator support 14 (23.7%) expired.

OPERATED AND NON-OPERATED CASES

Status of operation	Operated	Non-operated	Total
Survivors	27	26	53
Non-survivors	21	26	47
Total	**48**	**52**	**100**

Table 9 : Operated And Non-Operated Cases

GRAPHIC REPRESENTATION

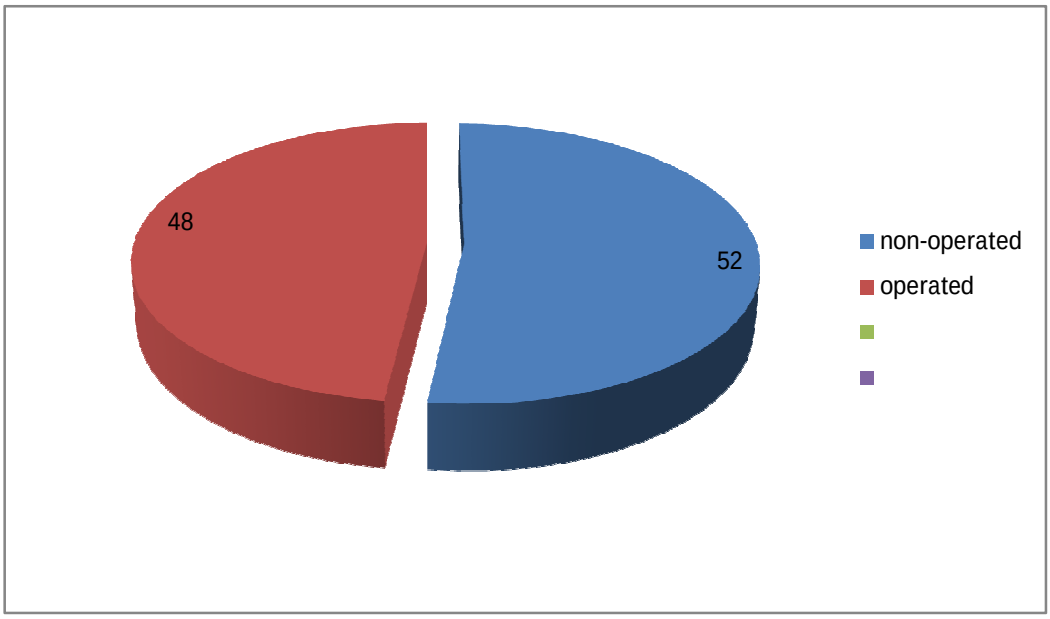

Figure 28: Comparison between Operated and Non-Operated Cases

STATUS OF BODY FLUID CULTURES

Positive	45
Negative	55

Table 10 : Status of Body Fluid Cultures

GRAPHICAL REPRESENTATION

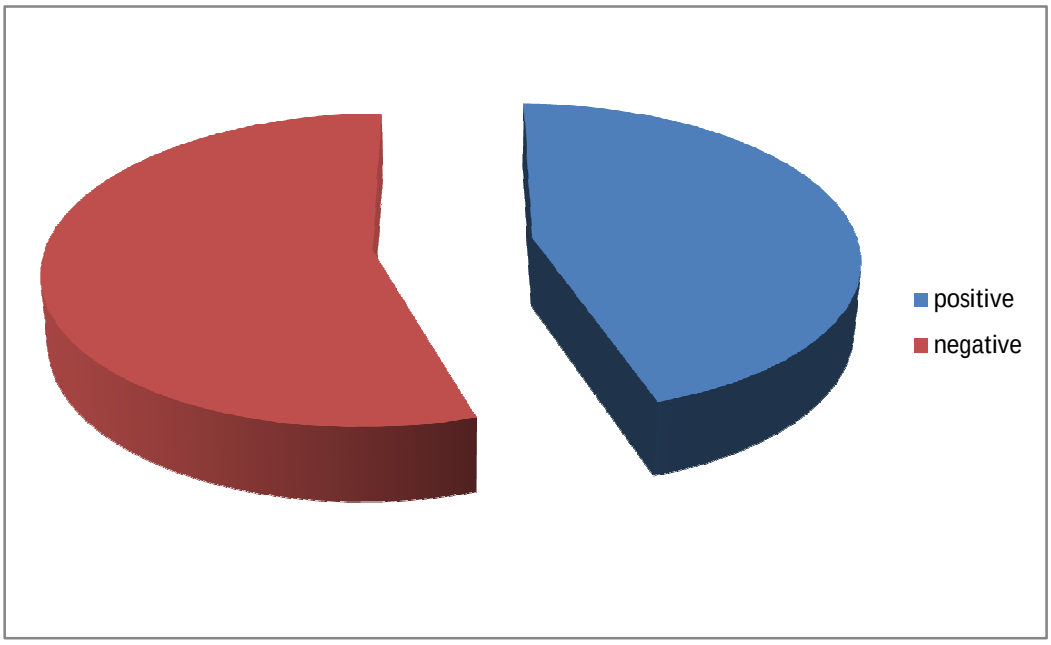

Figure 29 : Comparison between Status of Body Fluid Cultures

DISCUSSION

Since the cost of health care is increasing day to day, assessment of a patient's prognosis is vital during the course of treatment. Outcome prediction gains importance in this regard. So scoring systems have been used to predict this. SOFA scoring system, because of its simplicity and easy applicability, has been widely used in critical situation. This system has also been evaluated in many ICUs and found to be useful as a simple bedside tool.

In our study sex of the patient did not play a significant role in influencing mortality. The morbidity and mortality is purely related to the underlying disease state.

But, the need for mechanical ventilation clearly predicted mortality outcome, since the patients who were ventilated showed a higher mortality rate compared to those who did not require ventilator support, as evidenced by the statistically significant p value < 0.001.

There is a significant increase in mortality rate when the SOFA score is above 12. There is a steep rise in the mortality curve at this value. Admission SOFA, 48 hours SOFA and 96 hours SOFA are all statistically significant with a p value < 0.001

Delta SOFA which is the difference in values over a period of time is also statistically significant in our study. There is astrong evidence that,

patients whose delta SOFA values when increased from the previous value, there is a greater chance that the patient may succumb to his illness.

Mean SOFA value also proved to be an independent predictor of mortality. A value of more than 11 showed a sharp rise in mortality.

Total SOFA score is also statistically significant in predicting mortality, irrespective of the disease state. A total SOFA score of more than 33.5 is associated with increased mortality.

CONCLUSION

- SOFA score is very useful in predicting mortality in critically ill patients, since there is a strong correlation between a rise in the score and mortality in all stages of admission.
- Mechanically ventilated patients have a high risk of mortality compared to non ventilated patients.
- The total SOFA and Mean sofa are better predictors of mortality.
- Delta SOFA score is also a better predictor of mortality.
- Early prediction of outcome in sepsis using SOFA score is useful to aid suitable modification of management strategies.
- In our study, out of 51 patients whose admission SOFA score was very high (above 12), 16 patients were survived .This data depicts that, with early prediction of outcome using SOFA score and suitable therapeutic intervention, 16 critically ill patients were survived .
- Same way out of 32 pts whose SOFA score on admission was low (less than 8), 8 pts died. This data depicts, even with low SOFA score on admission, few patients died, because so many other factors are also contributing to the death of critically ill patients.
- So using SOFA scoring we can improve the overall prognosis and prevent the mortality to some extent.

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ANNEXURE – I

PROFORMA

I. Basic Details:

Name of the patient :

Age (in years) :

Sex : M / F

Diagnosis :

Surgery :

II. History:

i. Chief Complaints

ii. Duration

iii. History of present illness

iv. Past History

   a. History of Pulmonary Disease

   b. History of Diabetes

   c. History of Heart Disease

   d. History of previous surgery

v. Personal History

   a. History of smoking / alcoholism

III. General Examination:

Pulse rate :

Blood Pressure :

Respiratory Rate :

SPO2(saturation) :

Temperature :

Glassgow coma score :

IV. Systemic Examination:

i. Cardiovascular system

ii. Respiratory system

iii. Per abdomen examination

iv. Central nervous system

V. Blood investigations

I Serum creatinine

II Serum bilirubin

III Platelet count

VI.Follow up of patients

SOFA SCORE

	0	1	2	3	4
Respiration PaO2/FIO2 (mmHg) SaO2/FIO2	>400	<400 221–301	<300 142–220	<200 67–141	<100 <67
Coagulation Platelets 103/mm3	>150	<150	<100	<50	<20
Liver Bilirubin (mg/dL)	<1.2	1.2–1.9	1.9 2.0–5.9	6.0–11.9	>12.0
Cardiovascular Hypotension	No hypotension	MAP <70	Dopamine </=5 or Dobutamine (any)	Dopamine >5 or norepinephrine </=0.1	Dopamine >15 or norepinephrine >0.1
CNS Glasgow Coma score	15	13-14	10-12	6-9	<6
Renal Creatinine (mg/dL) or urine output(ML/dl)	<1.2	1.2–1.9	2.0–3.4	3.5–4.9 or <500	>5.0 or <200

MAP, mean arterial pressure; CNS, central nervous system; SaO2, peripheral arterial oxygen saturation. PaO2/FIO2 ratio was used preferentially. If not available, the SaO2/FIO2 ratio was used; vasoactive mediations administered for at least 1 hr (dopamine and norepinephrine ug/kg/min).

ANNEXURE - II

INFORMED CONSENT

DEPARTMENT OF GENERAL SURGERY

Coimbatore Medical College, Coimbatore

I have been invited to participate in the research project titled "*use of SOFA(SEQUENTIAL ORGAN FAILURE ASSESSMENT) scoring in assessing the incidence and severity of organ dysfunction and predicting the outcome in patients with sepsis in surgical unit"*

I understand, it will be answering a set of questionnaire, undergo physical examination, investigations and appropriate treatment. I also give consent to utilise my personal details for study purpose and can be contacted if necessary. I am aware that I have the right to withdraw at any time which will not affect my medical care.

Name of the participant :

Signature :

Date :

ANNEXURE - III

MASTER CHART

S.NO	Name	AGE	SEX	DIAGNOSIS	SOFA SCORE ON ADMISSION	SOFA SCORE AT 48 HOURS	SOFA SCORE AT 96 HOURS	OUTCOME	VENTILATOR SUPPORT	OPERATED
1	MURUGESAN	45	M	PERFORATED PERITONITIS WITH SEPTICAEMIA	9	11	15	B	YES	YES
2	VIJAYA	39	F	PERFORATED PERITONITIS WITH SEPTICAEMIA	6	6	4	A	NO	YES
3	LAKSHMI	40	F	ELEPHANT ATTACK	8	10	13	B	YES	NO
4	HAESHAN	35	M	PERFORATIVE PERITONITIS	10	12	10	A	NO	YES
5	KUMAR	65	M	ILEAL PERFORATION	12	16	18	B	NO	YES
6	YANISHA	19	F	INTRACRANIAL SOLITARY LESION	10	12	12	B	YES	NO
7	RAMAKRISHNAN	42	M	DUODENAL PERFORATION	8	14	14	B	YES	YES
8	BALAMANI	60	F	SIGMOID VOLVULUS	12	10	10	A	YES	YES
9	MANIKKAM	70	M	CARCINOMA STOMACH	13	15	15	B	YES	YES
10	SUMATHI	40	F	60% THERMAL BURNS	16	20	20	B	NO	NO
11	SELVAM	57	M	POSTAPPENDICECTOMY SEPTICEMIA	8	6	6	A	NO	YES
12	GOVINDARAJ	71	M	GASTRIC PERFORATION	8	4	4	A	NO	YES
13	AMUDHA	40	F	30% BURNS WITH SEPTICEMIA	6	6	6	A	YES	NO
14	JENCY	52	F	CELLULITIS LT LOWER LIMB	8	10	10	A	NO	YES
15	KANNAN	38	M	BLUNT INJURY ABDOMEN	15	18	20	B	NO	YES
16	SUBRAMANYAN	35	M	STAB INJURY WITH LIVER LACERATION	8	6	6	A	NO	YES
17	DEVRAJ	74	M	SMALL BOWEL PERFORATION	15	18	18	B	NO	YES
18	KARUPPASAMY	50	M	RUPTURED LIVER ABSCESS	12	10	10	A	NO	YES
19	KRISHNAN	63	M	ACUTE INTESTINAL OBSTRUCTION	10	6	6	A	NO	YES
20	VALLIYAMMAL	45	F	85% MIXED DEGREE BURNS	8	10	10	B	YES	NO
21	SELVAM	70	M	CAECAL PERFORATION WITH PERITONITIS	8	14	20	B	YES	YES

22	SANGEETHA	17	F	50% BURNS	12	16	16	B	YES	NO
23	SIVAIKUMAR	28	M	PERFORATIVE PERITONITIS	10	8	8	A	NO	YES
24	MURUGESH	40	M	ACUTE MESENTRIC ISCHEMIA	12	10	9	A	NO	YES
25	KITTUSAMY	50	M	SMALL BOWEL GANGRENE	8	6	6	A	NO	YES
26	RAMASAMY	65	M	ILEAL PERFORATION	12	10	10	B	NO	YES
27	KANNARAJAN	65	M	PERFORATIVE PERITONITIS	10	14	16	B	NO	YES
28	NANJAMMAL	70	F	40% MIXED DEGREE BURNS	8	10	8	A	NO	NO
29	MARUGATHAM	47	F	CELLULITIS WITH GANGRENE LT UPPER LIMB	11	10	10	A	NO	NO
30	SUBBATHAL	60	F	DIABETIC ULCER FOOT	14	12	12	A	NO	NO
31	BHUAMAN	59	M	CARCINOMA RECTUM WITH SECONDARIES	16	18	19	B	NO	NO
32	HYDHARALI	59	M	DIABETES WITH MULTIORGAN FAILURE	15	20	20	B	YES	NO
33	GOWRISANKAR	68	M	GANGRENE FOOT	12	16	18	B	YES	NO
34	CHANDRAN	41	M	PERFORATIVE PERITONITIS	13	10	10	A	NO	YES
35	KRISHNAVENI	50	F	DIABETIC ULCER FOOT	15	12	12	A	NO	NO
36	RANGASAMY	75	M	CELLULITIS RT UPPER LIMB	11	10	10	A	YES	NO
37	JAYARAM	60	M	GANGRENE FOOT	11	15	15	B	YES	NO
38	THNGAVEL	66	M	CELLULITIS RT LOWER LIMB	8	8	8	A	NO	NO
39	GOWRIYAMMAL	52	F	CELLULITIS RT LOWER LIMB	8	10	8	A	NO	NO
40	MOHAMMED	70	M	CELLULITIS RT FOREARM	12	10	10	A	NO	NO
41	IRUDHAYA RAJ	55	M	DIABETIC FOOT LT LOWER LIMB	8	6	6	A	NO	YES
42	BALU	37	M	30%BURNS	12	10	12	A	YES	NO
43	MICHIYAMMAL	74	F	PERFORATIVE PERITONITIS	12	13	13	B	YES	YES
44	RAJASEKAR	35	M	30% BURNS	13	10	10	A	YES	NO
45	VINOTH	54	M	GANGRENE RT FOOT	10	12	14	B	YES	NO
46	SARAVANAN	25	M	PERFORATIVE PERITONITIS	10	8	8	A	NO	YES
47	RAMESH	46	M	CARCINOMA PANCREAS	15	15	16	B	YES	NO
48	RAHUMAN	70	M	BLUNT INJURY ABDOMEN	13	14	14	B	NO	YES

49	LAKSHMI	50	F	OBSTRUCTED INCISIONAL HERNIA WITH FECAL FISTULA	13	14	16	B	YES	YES
50	PRADEEP	50	M	CELLULITIS RT LOWER LIMB	8	6	6	A	NO	NO
51	GUNASEKAR	60	M	60% ELECTRICAL BURNS	12	16	16	B	YES	NO
52	NOORJAHAN	53	F	DIABETIC FOOT LT LOWER LIMB	8	7	7	A	NO	NO
53	NATARAJAN	55	M	NECROTIZING FASCITIS RT LOWER LIMB	14	16	17	B	YES	NO
54	SANGUMUTHU	40	M	SMALL BOWEL GANGRENE	10	9	8	A	NO	YES
55	RAVISANKAR	52	M	CELLULITIS LT LOWER LIMB	16	16	18	B	YES	NO
56	GOVINDAN	66	M	GANGRENE RT FOOT	13	12	12	B	YES	NO
57	MANISHARMA	56	M	PERFORATIVE PERITONITIS	14	15	16	B	YES	YES
58	LAKSHMI	43	F	DIABETIC FOOT LT LOWER LIMB	11	10	10	A	NO	NO
59	SELVARAJ	46	M	FOURNIERS GANGRENE	16	19	20	B	YES	NO
60	BAKKIAMMAL	80	F	DIABETIC FOOT WITH COPD	13	10	10	A	NO	NO
61	INBARAASU	72	M	HEPATOCELLULAR CARCINOMA	8	8	9	B	NO	NO
62	ELANGOVAN	46	M	BLUNT INJURY ABDOMEN	8	6	6	A	NO	YES
63	PALANIYAMMAL	75	F	CELLULITIS BOTH LOWER LIMBS	10	8	8	A	YES	NO
64	ANBUMANI	61	M	SNAKE BITE CELLULITIS	8	10	10	A	NO	YES
65	SIVASENBAGAM	40	F	GANGRENE RT FOOT	12	12	10	B	NO	NO
66	SUNDAR	45	M	80% BURNS	16	12	12	B	YES	NO
67	HARIHARAN	35	M	30% BURNS	8	7	7	A	NO	NO
68	BATHRAN	60	M	DIABETIC ULCER LT LOWER LIMB	8	6	6	A	NO	NO
69	PALANISAMY	65	M	DIABETIC GANGRENE LT LOWER LIMB	12	16	16	B	YES	NO
70	MARIYAPPAN	66	M	CELLULITIS LT LOWER LIMB	7	10	9	A	NO	YES
71	LAKSHMI	45	F	NECROTIZING FASCITIS RT LOWER LIMB	16	18	18	B	YES	NO
72	SATHISH	30	M	STRANGULATED INGUINAL HERNIA	20	22	22	B	YES	YES
73	KANDHASAMY	60	M	INTESTINAL OBSTRUCTION WTH BOWEL GANGRENE	10	6	6	A	NO	YES
74	SULOCHANA	63	F	DIABETIC ULCER RT FOOT	16	12	12	A	NO	NO
75	NAGARATHINAM	60	F	RT BREAST ABSCESS	20	18	18	B	YES	YES

76	AYYASAMY	49	M	SMALL BOWEL GANGRENE	16	18	18	B	N0	YES
77	SUSEELA	45	F	DIABETIC KETOACIDOSIS WITH CELLULITIS	6	6	6	A	NO	NO
78	UMADEVI	60	F	CELLULITIS BOTH LOWER LIMBS	12	14	14	A	NO	NO
79	RAMASAMY	60	M	ISCHEMIC BOWEL DISEASE	18	16	16	B	YES	NO
80	PANDIYAN	40	M	PNEUMOTHORAX	10	8	8	A	NO	YES
81	ABDUL RAHAMAN	53	M	CARCINOMA STOMACH	12	14	16	B	YES	NO
82	ANTONY MURTHY	75	M	PERFORATIVE PERITONITIS	13	12	12	A	NO	YES
83	MANIKANDAN	25	M	POLYTRAUMA-POST SPLEENECTOMY	13	16	18	B	NO	YES
84	MANILLAVARASAN	43	M	ILEOCOLIC INTUSUSSEPTION	12	12	8	A	YES	YES
85	MURUGESAN	45	M	GASTROJEJUNOSTOMY WITH STOMAL PERFORATION	8	8	6	A	NO	YES
86	SUBRAMANI	65	M	CARCINOMA RECTUM	14	14	16	B	YES	NO
87	RAMACHANDRAN	47	M	ILEAL PERFORATION	8	8	8	B	YES	YES
88	RAJAN	60	M	MESENTERIC VASCULAR ISCHAEMIA	13	13	12	B	NO	YES
89	SELVARAJ	59	M	HEPATOCELLULAR CARCINOMA	6	8	9	B	YES	NO
90	THANGAMMAL	58	F	NON-HEALING ULCER LT FOOT	12	12	13	B	YES	NO
91	THIRUMOORTHY	50	M	BOWEL ISCHAEMIA	14	12	12	A	NO	NO
92	PRIYADHARSHINI	23	F	SNAKE BITE CELLULITIS	8	6	6	A	NO	NO
93	MUNUSAMY	63	M	PERFORATIVE PERITONITIS	10	10	9	A	NO	YES
94	RAMACHANDRAN	35	M	RUPTURED LIVER ABSCESS	13	12	13	B	YES	YES
95	PHILOMINA	65	F	DIABETIC FOOT RT	9	10	10	A	NO	NO
96	SAROJA	70	F	DIABETIC FOOT RT	14	13	13	A	YES	NO
97	ARUNKUMR	20	M	PERFORATIVE PERITONITIS	10	12	13	A	NO	YES
98	GOPAL	63	M	CARCINOMA RECTOSIGMOID	16	16	18	B	NO	YES
99	LAKSHMI	45	F	CELLULITIS LT LOWER LIMB	6	7	7	A	NO	NO
100	KUMAR	38	M	ILEAL PERFORATION	8	8	7	A	NO	YES

A – SURVIVORS, B – NON SURVIVORS